THE PLANT DISEASE REPORTER

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PLANT DISEASE EPIDEMICS
and
IDENTIFICATION SECTION

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PLANT DISEASE EPIDEMICS AND IDENTIFICATION SECTION

Horticultural Crops Research Branch — Plant Industry Station, Beltsville, Maryland

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SOME VIRUSES OF THE FIELD TOMATO IN ONTARIO

Blair H. MacNeill

INTRODUCTION

Under the sponsorship of the Research Council of Ontario a survey of the mechanically transmitted viruses of the field tomato was carried out during the season of 1952 and 1953. This survey covered the general areas of canning crop production in Ontario, although sampling was most intensive in the counties of Prince Edward, Northumberland, Essex and Kent. Areas which were not covered by personal survey were, in most instances, checked by commercial fieldmen, professional staff of Science Service laboratories, and in many cases the growers themselves, who collected sample materials and forwarded them to us for analysis.

PROCEDURE

In analysing the samples of tomato foliage and fruit we used as our reference the work of Ainsworth, Berkeley and Caldwell (1), who in 1934 had made a comparison of the English and Canadian tomato virus diseases. In anticipation of a situation similar to that which they reported, the following array of differential host material was used:

Lycopersicum esculentum Mill var. Bonny Best
Nicotiana tabacum L. var. Harrow Velvet
N. glutinosa L.
N. rustica L.
Cucumis sativus L. var. A & C
Datura stramonium L.
C. album L.
Gomphrena globosa L.
Vigna sinensis Endl. var. Black
Zinnia elegans Jacq.
Capsicum frutescens L.
Physalis peruviana L.
P. floridana L.

RESULTS

During this survey approximately 200 samples were examined for virus content. In all cases, juice from the samples was extracted mechanically in a mortar containing a buffer solution of disodium hydrogen phosphate, and then the plants, previously dusted with carborundum, were inoculated by rubbing with the extract. From this survey, using data based on 1) the differential reactions of the hosts listed above, 2) thermal inactivation tests, and 3) cross-protection and serological reactions, it was concluded that at least six viruses or combinations of viruses were present in the tomato crop. These viruses, together with the symptoms which they commonly produce on tomato, are listed in Table 1.

Nicotiana virus 1 (TMV), which generally gives only a mild mottle on tomato with little or no distortion, has proved to be the virus most commonly encountered. At the same time, isolations of a severe distortion type, which produces symptoms which may be confused with injury arising from substances like 2, 4-D, have not been uncommon. Undoubtedly, TMV is the most common virus to be found on the tomato, both before the transplanting of the seedlings and after the plants have been set in the field.

The appearance of the streak symptom in the field has been found to be due to the action of at least three viruses or combinations of viruses. This symptom is of very general occurrence and has usually been attributed to Lycopersicum virus 1, which itself seems to be a strain of TMV. For the most part, however, the streak that appears from mid-season through to harvest we have found to be of the double-virus type and to result from the combined action of TMV and Solanum virus 1 (Potato X). It is significant that the TMV component, when freed from this streak complex, is exceedingly mild in its reaction on tomato.

The etch symptom, arising from infection by Nicotiana virus 7, is easily overlooked in the field, as there is present frequently only a faint mottle or a slight curl. Under experimental conditions in the greenhouse, our etch strain has been found to induce in the tomato a tendency toward spindly foliage and poor fruit set. The distribution of the virus appears to be confined

Table 1. Viruses obtained by juice extraction from foliage and fruit of the field tomato.

Virus	Type of symptom on tomato
Nicotiana virus 1	Mottle
Lycopersicum virus 1	Streak
Nicotiana virus 7	Mottle and necrotic flecking
Solanum virus 1 in combination with Nicotiana virus 1	Streak
Lycopersicum virus 3	Spotted wilt
Solanum virus 2 in combination with Nicotiana viruses 1 and 7	Streak--variable

to the western part of the Province, and as this area is the one where imported seedlings are rather generally used it was suggested that plants coming in from the Southern United States were supplying the etch inoculum each season. However, careful sampling of such plants at ports of entry have given completely negative results. Presumably the plants come in contact with the virus sometime after they are set in the field.

Cucumber mosaic virus has been isolated from samples only infrequently. Characteristically, some strains produce the familiar "shoestring" symptom, although this reaction is often the result of TMV infection. For the most part, the cucumber mosaic virus strains that have been isolated are very mild on tomato, and the symptoms may be readily overlooked in the field.

To date, two instances of infection with the tomato spotted wilt virus have come to our attention. These appeared in two successive years in a field in the Fort Erie district; there was no evidence that the disease was widespread. However, the virus is not readily transmitted mechanically, and negative results from samples suspected of being infected with the spotted wilt virus may not be altogether reliable.

DISCUSSION

Undoubtedly, the tobacco mosaic virus is very important as a disease agent in the tomato crop. Acting either alone or in combination with several other viruses, it causes widespread destruction; by mid-season it is present in almost every commercial tomato field. The prevalence of TMV possibly may be explained by the fact that tomato plants are handled a great deal during their culture, and a virus as persistent as TMV is almost certain to become widely distributed. Nevertheless, it is difficult to understand how, in many cases, over 85 percent of the plants in several contiguous fields of a district could become infected in the absence of an insect vector.

The very common occurrence of the etch disease perhaps is to be expected, since etch, according to several workers, appears to be on the increase in tobacco (4) in southern Ontario, and now is prevalent on peppers (3) throughout this same district. Certainly, it is not difficult to find tomatoes infected with the etch virus, although whether this strain is the same as appears on peppers and tobacco is not known. The tomato strain is not severe on the tomato host and its action on Burley tobacco is likewise very mild. However, there is considerable evidence that the etch virus drastically reduces the yield of the tomato by inducing flower drop, and this insidious action may be responsible for as much crop loss as results from a more conspicuous virus such as TMV.

Of all the virus diseases of the tomato, streak is the most conspicuous in the field; undoubtedly it accounts for the major part of the visible crop loss. The consistent association of Solanum virus 1 (Potato X) in this double-virus complex poses the problem of the origin and

distribution of a virus that is not generally insect-borne. Field observations suggest that potatoes in nearby plots increase the incidence of double-virus streak, as does the planting of tomatoes in fields which produced potatoes the previous season. Also, Solanum virus 2 (Potato Y) has appeared in several isolations from the streak complex; this finding is in agreement with a recent report (2) from Florida in which Potato Y was said to occur on tomato either alone or in combination with TMV. Further, the presence of necrotic strains of TMV, which alone may produce a streaking of the stems of the tomato, so complicates the picture that a field diagnosis of the streak symptom can be anything but reliable.

The infrequency with which we encountered the so-called typical strain of Cucumis virus 1 was not to be expected from the reports in the literature. The shoestringing effect, commonly ascribed to the cucumber mosaic virus, almost invariably could be traced to a strain of TMV, and only in a few instances was Cucumis virus 1 obtained alone from such material. Possibly the method of juice extraction was responsible for the large proportion of negative results. However, certain viruses which we believe to be strains of the cucumber mosaic virus have been obtained; these are, for the most part, very mild on tomato but exceedingly severe on cucumber -- so severe in fact that the virus cannot be maintained on this host, the plants dying within 10 days of inoculation. Whether these isolates are genuine strains of Cucumis virus 1 it is not possible to say at this time, yet they appear to fall within the orbit of this virus as determined by host reaction, thermal inactivation, and cross-protection tests with Price's local lesion strain. Obviously, this whole complex of entities, collectively known as cucumber mosaic virus, needs to be very carefully worked over and redescribed with perhaps new and more useful criteria. Such as an analysis of "cucumber mosaic" in the tomato would likely yield some results of fundamental interest to the plant virologist.

While this survey has dealt only with those viruses which may be mechanically transmitted from the tomato, there is little evidence that viruses which are exclusively insect-borne are of great significance in the Ontario canning districts. Yet the role of insects in spreading many of the viruses noted above must be considered, especially in view of cultural practices that place related susceptible crops such as potatoes, peppers, tobacco, and tomatoes in contiguous locations and that permit the widespread persistence of weeds which may act as reservoirs for the tomato viruses.

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DEPARTMENT OF BOTANY, ONTARIO AGRICULTURAL COLLEGE, GUELPH, ONTARIO

HOST RANGE AND VARIABLE PATHOGENESIS OF THE NECROTIC RING SPOT VIRUS IN THE GENUS PRUNUS[1]

R. M. Gilmer

Summary

Immunity to necrotic ring spot was not found in any of 122 species of Prunus tested, representing all botanical sections of the genus, and it was concluded that the probability of the occurrence of immunity to this disease within the genus is slight. Varying degrees of tolerance to necrotic ring spot occurred in certain Prunus species, none of which are closely enough related to the true cherries to indicate that the incorporation of such tolerance into commercial varieties would be practicable.

Seven isolates containing necrotic ring spot virus alone or in combination with other viruses gave an aggregate severity rating, when inoculated into a group of eight Prunus species, that was essentially the same for each isolate. All isolates incited a typical syndrome in Prunus and in cucumber, and these syndromes were considered to be incited by the necrotic ring spot virus.

The possibility that necrotic ring spot virus is an essential component of the sour cherry yellows complex is discussed.

INTRODUCTION

A disease of plum and peach similar to if not identical with necrotic ring spot was described as early as 1932 (6), but the first incontestable description of the disease was made in 1939 and its virus etiology was demonstrated shortly thereafter (8). The causal virus is widely distributed and extremely prevalent in sour and sweet cherry plantings in the United States and Canada and occasionally has been found in other species of stone fruits. Evidence of economic damage is somewhat conflicting (1), but, in general, necrotic ring spot is regarded as a disease of relatively minor importance. Although necrotic ring spot virus per se may be unimportant, it is apparently a frequent, if not universal, concomitant of the sour cherry yellows virus complex which is of great economic importance.

The present nursery indexing program for the production of virus-free stone fruits in New York and other States relies heavily upon the identification of necrotic ring spot virus and its elimination from propagating clones, since the elimination of ring spot infected trees apparently also eliminates those infected with sour cherry yellows. However, eastern sour cherry orchards, when planted with virus-free nursery trees, are characteristically soon attacked by necrotic ring spot and, sequentially, by sour cherry yellows (3, 4, 5).

Considerable uncertainty exists regarding the association of necrotic ring spot virus and the sour cherry yellows complex, i. e., whether necrotic ring spot virus is an essential component of the sour cherry yellows complex or whether its almost universal association with this complex is merely fortuitous. Solution of this uncertainty may prove of considerable practical importance, since it has been informally suggested that one means of preventing the rapid spread of necrotic ring spot in orchard plantings might be the intentional inoculation of nursery trees prior to planting with a "mild" strain of the virus. Such prior infection might protect the trees against the advent of more "severe" forms of the disease and the accompanying decrease in yields during its acute stage, but whether such inoculation might not also increase the rate of spread of cherry yellows is problematical.

A second obvious approach to the problem of orchard spread of necrotic ring spot is the detection of Prunus species immune from the causal virus. Such species could be employed as a source of immunity for incorporation into stone fruit clones or understocks by means of a breeding program, and, additionally, would possess considerable scientific interest as a means of isolating components of the sour cherry yellows virus complex free of necrotic ring spot virus.

The purpose of the present investigation was to discover whether immunity from necrotic ring spot virus exists in the genus Prunus and to determine whether the necrotic ring spot virus

[1] Approved by the Director for publication as Journal Paper No. 988, New York Agricultural Experiment Station, Geneva, New York, January 19, 1955.

Table 1. Source, region of origin, and probable virus content of seven isolates containing necrotic ring spot virus.

Source	Region of origin	Virus content
CR 5-1	Geneva, New York	NRS[a] + Y + PD
Lewis 10-10	Geneva, New York	NRS + Y + ?
B7/8-1[b]	Sodus, New York	NRS
A10/1-5[b]	Sodus, New York	RNRS + Y
Barnard 1-12[c]	Sturgeon Bay, Wisconsin	NRS
Barnard 3-22[c]	Sturgeon Bay, Wisconsin	NRS + Y + PD
S-5009[c]	Sturgeon Bay, Wisconsin	RNRS + Y

[a] NRS represents a strain of necrotic ring spot virus latent in Montmorency sour cherry after initial acute symptoms; RNRS, a strain of the same virus that incites striking annually-recurring symptoms in the same host; Y, sour cherry yellows virus; PD, prune dwarf virus.

[b] Supplied by Dr. K. G. Parker, Cornell University.

[c] Supplied by Dr. J. Duain Moore, University of Wisconsin.

is an essential component of the sour cherry yellows complex.

MATERIALS AND METHODS

All of the host range tests were made in the greenhouse and almost all were carried out in the period January-May under automatically controlled temperatures, 75° F during the day (8 hours) and 55° F at night (16 hours). A few tests were made in August and September when close temperature control in the greenhouse could not be attained.

Approximately 122 species or subspecies of Prunus[2] were included in the host range studies. Most of the individual species were maintained as clones, but a few species were grown from seeds.

Inoculations were made at or immediately after budbreak by patch-budding the test trees with two buds of Montmorency cherry or Lovell peach infected with the appropriate virus isolate. At least three trees of each species were inoculated with each virus isolate, and at least four, and frequently seven, isolates were tested on each species. In certain of these isolates only necrotic ring spot virus was present, while in others either cherry yellows virus or cherry yellows and prune dwarf viruses were present. The virus isolates, the location from which they were obtained, and their probable virus content are given in Table 1.

Each of the isolates was initially tested for transmission of a virus entity to cucumber seedlings (var. Chicago Pickling). No consistent differences were evident in type of symptoms incited in cucumber seedlings inoculated with the various isolates and all incited an apparently identical disease.

Routine reisolations from all inoculated Prunus plants were made to mazzard or Lovell peach seedlings. No attempts were made to determine whether viruses other than that of necrotic ring spot were recoverable from the experimentally inoculated trees. Without significant exception, the reisolations incited a disease of Lovell peach or mazzard cherry apparently identical with necrotic ring spot.

For the comparative study of the effects of the seven virus isolates on eight Prunus species especially selected for their differential response to necrotic ring spot virus, each of the eight species was propagated from a single clone. Each virus isolate was tested on ten budlings of each species in 1953 and the test was repeated in 1954. The individual budlings were random-

[2] Principal sources were the Arnold and Morton Arboretums and the U. S. Plant Introduction Garden, Chico, California. Many European and Asiatic species were obtained from Hillier and Sons, Winchester, England, through the courtesy and cooperation of Dr. J. L. Creech, U. S. Plant Introduction Garden, Glenn Dale, Maryland.

Table 2. Infection of various Prunus species by isolates containing necrotic ring spot virus, 1953-54.

Species	5-1	7/8-1	10-10	1-12	3-22	5009	10/1-5
alabamensis	+[a]	+	+	. .	. .	. .	+
alleghaniensis	+	+	+	+	+	+	+
americana	+	+	+	+	+	+	+
andersoni	+	+	+	+	+	+	+
angustifolia	+	+	+	. .	. .	. .	+
apetala	+	+	+	. .	. .	+	+
armeniaca	+	+	+	+	+	+	+
avium	+	+	+	+	+	+	+
besseyi	+	+	+	+	+	+	+
blieriana	+	+	+	. .	. .	+	. .
bokharensis	+	+	+	. .	. .	. .	+
bucharica	+	+	+	. .	. .	. .	+
campanulata	+	+	+	. .	. .	. .	+
canescens[b]	+	+	+	. .	. .	. .	+
caroliniana	+	+	+	+	+	+	+
cerasifera	+	+	+	+	+	+	+
cerasoides	+	+	+	. .	. .	. .	+
cerasus	+	+	+	+	+	+	+
cistena[c]	0	0	0	0	0	0	0
communis	+	+	+	. .	. .	. .	+
conradinae	. .	. .	+	+	+	+	. .
cornuta	+	+	+	. .	. .	. .	+
cyclamina	+	+	+	. .	. .	. .	+
dasycarpa	+	. .	+	+	. .	. .	+
davidiana	+	+	+	+	+	+	+
demissa	+	+	+	+	+	+	+
dielsiana	+	+	+	. .	. .	. .	+
domestica	+	+	+	+	+	+	+
dropmoreana	+	+	+	+	+	+	+
dunbari	+	+	+	. .	. .	. .	+
effusa	+	+	+	+	+	+	+
emarginata	+	+	+	. .	. .	. .	+
fasciculata	+	+	+	. .	. .	. .	+
fenzliana	+	+	+	+	+	+	+
fontanesiana	+	+	. .	. .	. .	+	. .
fremonti	+	+	+	. .	. .	. .	+
fruticosa	+	+	+	+	+	+	+
gigantea	+	+	+	. .	. .	. .	+
glandulifolia	+	+	+	. .	. .	. .	+
gracilis	+	+	+	. .	. .	. .	+
glandulosa	+	+	+	+	+	+	+
gravesi	+	+	+	. .	. .	. .	+
grayana	+	+	+	. .	. .	+	. .
hillieri	+	+	+	+	+	+	+

Table 2. continued.

Species	5-1	7/8-1	10-10	1-12	3-22	5009	10/1-5
hortulana	+	+	+	+	+	+	+
ilicifolia	+	+	+	+	+	+	+
incana	. .	. .	. .	+	+	+	+
incisa	+	+	+	. .	. .	. .	+
insititia	+	+	+	. .	. .	. .	+
jacquemonti	+	+	+	. .	. .	+	+
japonica	+	+	+	+	+	+	+
juddi	+	+	+	. .	. .	. .	+
kansuensis	+	+	+	. .	. .	. .	+
kurilensis	+	+	+	. .	. .	. .	+
lanata	+	+	+	. .	. .	. .	+
lannesiana	+	+	+	. .	. .	. .	+
laucheana	+	+	+	. .	. .	. .	+
laurocerasus	+	+	+	+	+	+	+
lobulata	+	+	+	. .	. .	. .	+
lusitanica	. .	+	+	. .	+	. .	+
lycioides	+	+	+	. .	. .	. .	+
lyoni	+	+	+	+	+	+	+
maacki	+	+	+	+	+	+	+
mahaleb	+	+	+	+	+	+	+
mandshurica	+	+	+	. .	. .	. .	+
maritima	+	+	+	+	+	+	+
maximowiczi	+	+	+	. .	. .	. .	+
mexicana	+	+	. .	. .	. .	. .	. .
meyeri	+	+	+	+	+	+	+
mira	+	+	+	. .	. .	. .	+
monticola	+	+	+	+	+	+	+
mugus[d]	0	0	0	. .	. .	0	0
mume	+	+	+	+	+	+	+
munsoniana	+	+	+	+	+	+	+
nana	+	+	+	+	+	+	+
newporti	+	+	+	+	+	+	+
nigra	+	+	+	. .	. .	. .	+
nipponica	+	+	+	. .	. .	. .	+
padus	+	+	+	+	+	+	+
pensylvanica	+	+	+	+	+	+	+
persica	+	+	+	+	+	+	+
petunnikowi	+	+	+	. .	. .	. .	+
pilosiuscula	+	+	+	. .	. .	. .	. .
pleiocerasus	+	+	+	. .	. .	+	. .
potanini	+	+	+	. .	. .	. .	+
pumila	+	+	+	+	+	+	+
reverchoni	+	+	+	. .	. .	. .	+
rufa	+	+	+	. .	. .	. .	+
salicina[e]	+	+	+	+	+	+	+
sargenti	+	+	+	. .	. .	. .	+

Table 2. Continued.

Species	5-1	7/8-1	10-10	1-12	3-22	5009	10/1-5
scopulorum	+	+	+	. .	. .	. .	+
serotina	+	+	+	+	+	+	+
serrula	+	+	+	. .	. .	. .	+
serrulata	+	+	+	+	+	+	+
setulosa	+	+	. .	. .	+	. .	+
sibirica	+	+	+	. .	. .	. .	+
sieboldi	+	+	+	+	+	+	+
skinneri	+	+	+	+	+	+	+
simoni	+	+	+	. .	. .	. .	+
slavini	+	+	+	. .	. .	. .	+
spinosa	+	+	+	+	+	+	+
ssiori	+	+	. .	. .	+	+	. .
subcordata	+	+	+	. .	. .	. .	+
sultana	+	+	+	. .	. .	. .	+
subhirtella	+	+	+	+	+	+	+
tenella	+	+	+	+	+	+	+
tomentosa	+	+	+	+	+	+	+
triloba	+	+	+	. .	. .	+	+
umbellata	+	+	+	. .	. .	. .	+
ussuriensis[f]	+	+	+	+	+	+	+
ursina	+	+	+	. .	. .	. .	+
venulosa	+	+	+	. .	. .	. .	+
virginiana	+	+	+	+	+	+	+
yedoensis	+	+	+	+	+	+	+

[a] + indicates symptoms expressed; 0, symptoms not expressed; . . indicates no test..

[b] One clone from the Morton Arboretum was naturally infected with necrotic ring spot.

[c] In agreement with Young's findings (10), this species did not show symptoms, but NRS virus was recovered to mazzard seedlings.

[d] The only available clone was infected with NRS (?) prior to inoculations; no additional reaction was seen following inoculation with the 5 isolates indicated.

[e] A total of 20 varieties and crosses of P. salicina were tested.

[f] Probably merely a subspecies or variety of P. salicina.

ized on the greenhouse bench and were individually graded on a scale of 0-4 which depended on the severity of symptom expression.

EXPERIMENTAL RESULTS

Host Range

Although varying degrees of severity of symptom expression were evident on different host plants, the symptoms expressed within 3 weeks of inoculation were surprisingly uniform, consisting of chlorotic or necrotic flecking, rings, or spots. Occasionally, oak leaf patterns were evident in the leaves of certain trees, but this symptom characteristically occurred in relatively few cases and was usually delayed until partial recovery from the acute stage of infection was evident. Typically, all plants of inoculated series showed an acute stage of the disease ac-

Table 3. Average symptom ratings of eight Prunus species inoculated with seven isolates of necrotic ring spot virus, 1953-54.

Species	Rating[a] - 1953	Rating - 1954
caroliniana	1.03 (1.0-1.2)[b]	1.09 (1.0-1.2)
persica	1.07 (1.0-1.2)	1.14 (1.0-1.4)
ilicifolia	1.11 (1.0-1.4)	1.13 (1.0-1.3)
glandulosa	2.17 (1.7-2.6)	2.23 (2.0-2.5)
cerasus	2.91 (2.6-3.2)	3.04 (2.7-3.3)
nana	3.10 (2.6-3.6)	3.01 (2.8-3.2)
serrulata	3.29 (3.0-3.5)	2.96-(2.6-3.2)
dropmoreana	3.50 (3.1-3.7)	3.34 (3.0-3.6)
F value	178.3**	221.9**
LSD (.01)	.31	.25

[a] Each of 10 trees was graded 0-4 dependent upon severity of symptoms. The rating given is the average individual tree rating. (0 = no symptoms; 1 = slight chlorotic spotting of a few leaves; 2 = necrosis of few to several leaves; 3 = leaf necrosis and limited terminal dieback; 4 = terminal dieback to previous annual growth).

[b] Limits of symptom severity for the species per 10-tree series.

Table 4. Average individual isolate ratings of seven sources of necrotic ring spot virus inoculated to eight Prunus species, 1953-54.

Isolate	Rating[a] - 1953	Rating - 1954
CR 5-1	2.48 (1.0-3.7)[b]	2.24 (1.0-3.5)
19B7/8-1	2.25 (1.0-3.7)	2.24 (1.0-3.3)
A10/1-5	2.25 (1.0-3.5)	2.28 (1.0-3.5)
Lewis 10-10	2.31 (1.0-3.7)	2.30 (1.1-3.2)
Barnard 1-12	2.12 (1.0-3.2)	2.22 (1.0-3.6)
Barnard 3-22	2.35 (1.1-3.5)	2.31 (1.1-3.4)
S-5009	2.15 (1.0-3.6)	2.12 (1.0-3.0)
F value	2.35*	NS
LSD (.05)	.21	--

[a] See footnote a, Table 3.
[b] See footnote b, Table 3.

companied by cessation of growth and symptoms of varying degrees of severity, followed by a recovered stage in which either new growth was symptomless or only mild symptoms occurred. In occasional host plants, such as P. glandulosa (Clone 5073) and P. laurocerasus (Clone 5107), symptoms occurred that were probably attributable to other viruses present in certain of the isolates, but these symptoms seldom appeared before completion of a dormant period following inoculation.

Because necrotic or chlorotic flecking or spotting appeared in all inoculated host plants irrespective of the virus isolate used in inoculation, this syndrome was attributed to the necrotic ring spot virus. Data on reaction to necrotic ring spot virus are given in Table 2.

Variable Pathogenesis of the Seven Virus Isolates

The experiments dealing with the differential response of 8 Prunus species to the seven virus isolates were repeated in 1954 with essentially the same results as found in the 1953 experiments. The data on individual species response to the seven virus isolates are given in Table 3.

Comparison of Table 3 with Table 4, which contains data on the aggregate severity rating of each of the virus isolates on the eight differential Prunus species, reveals that the severity of symptom expression was primarily dependent upon the species inoculated and not upon the individual isolate employed. Insofar as species reaction is concerned, the species tested represent three groups: Group I, in which symptoms are mild (P. caroliniana, P. persica, P. ilicifolia); Group II, in which symptoms were intermediate in severity (P. glandulosa); and Group III, in which severe leaf necrosis and terminal dieback were characteristic (P. cerasus, P. dropmoreana, P. serrulata, and P. nana). In regard to isolate reaction, no clear-cut differences in magnitude of symptom severity were observed, although two of the Wisconsin isolates, Barnard 1-12 (NRS only) and S-5009 (RNRS + Y), incited the least severe symptoms in both years.

DISCUSSION

None of the 122 species of Prunus, representing all botanical sections of the genus, was found immune from necrotic ring spot. Since comparatively few species of the genus were not tested, this fact affords strong presumptive evidence that immunity to this disease does not exist within the limits of the genus. Varying degrees of tolerance to necrotic ring spot were evident, however. In species such as P. caroliniana and P. ilicifolia, symptoms of necrotic ring spot were very mild and of short duration. Following the mild initial stage of the disease, recovery was rapid in these species. Since these species, and several others that reacted in a similar manner, are only distantly related to the true cherries (Section Eucerasus Koehne), it is doubtful whether an attempt to incorporate such tolerance into the true cherries is feasible.

Whether necrotic ring spot virus is an essential component of the sour cherry yellows complex cannot be determined from the data obtained in the present investigation. While no evidence inconsistent with this hypothesis was found, no evidence unconditionally supporting the hypothesis was obtained. However, the present data, and those reported elsewhere (2, 7, 9), favor the hypothesis rather than otherwise. Boyle et al. (2) found that of eight virus isolates obtained from sour cherry (two containing latent necrotic ring spot virus, four containing a latent necrotic ring spot virus and yellows virus, and two containing a recurrent necrotic ring spot virus and yellows virus) each incited a disease of cucumber following mechanical inoculation. Symptoms appearing in cucumber were quite similar for all of the various isolates (2). An apparently identical situation was found in the present study; all seven isolates (of which two, S-5009 and Barnard 3-22, were employed by Boyle et al. (2), irrespective of virus content, incited an essentially identical disease in cucumber.

Weintraub and Willison (7), dealing with stone fruit virus isolates infective to cucumber, reported that neither the thermal death point nor the temperature coefficient of thermal inactivation could be determined reproducibly for 13 virus isolates obtained from Prunus hosts infected with sour cherry yellows, necrotic ring spot, green ring mottle, tatter-leaf, and prune dwarf. All of their virus isolates incited a syndrome in cucumber apparently indistinguishable from that of Boyle et al. (2) and of the present study. However, in certain of their isolates (G. 1 and T. 2, obtained from trees infected with green ring mottle and tatter-leaf, respectively), two apparently distinct virus components were found (7). In a later paper (9), they reported that isolate Y. 2 (sour cherry yellows) was also partially separable into two components in cucumber. Their component B, found in these three isolates, incited a chlorotic ring spotting of

cucumber apparently identical with symptoms incited by necrotic ring spot virus as reported by Boyle et al. (2) and as found in the present investigation. These data (7, 9) are extremely suggestive that at least one virus component is common to all of their isolates, and coupled with the findings of Boyle et al (2) and of the present study, it may be argued that this component (component B) is the necrotic ring spot virus.

A comparison of the data of Boyle et al. (2) and of Willison and Weintraub (7, 9) with those of the present investigation shows that in no case in the total of 11 isolates containing sour cherry yellows virus was the latter found unaccompanied by an entity capable of inciting a disease of cucumber indistinguishable from the disease incited by necrotic ring spot virus. This fact strongly suggests that necrotic ring spot virus is present in all 11 yellows isolates obtained from three diverse geographic areas. Such constant association of necrotic ring spot and yellows viruses argues that necrotic ring spot virus is an essential component of the yellows complex.

The possibility that a virus other than necrotic ring spot virus may incite a syndrome in cucumber indistinguishable from that caused by necrotic ring spot virus is not unquestionably excluded by the foregoing discussion. However, all seven isolates employed in the present study, irrespective of their total virus content, incited a characteristic syndrome in all of the Prunus species tested as well as a typical syndrome in cucumber, a further indication that the two syndromes were incited by a single virus entity common to all of the isolates, the necrotic ring spot virus.

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AMMONIA GAS TO CONTROL BLUE-GREEN MOLD DECAY OF CITRUS FRUITS

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Tomkins and Trout (4) reported that damp crystals of ammonium bicarbonate in tissue with injured-inoculated oranges inhibited decay by green mold. They stated also that continuous exposure to 500-1000 ppm of gaseous ammonia secured by bubbling air through aqueous solutions of ammonia was sufficient to prevent decay and that a concentration of 2000 ppm was injurious to the fruit. Apparently because of the injury hazard they made no attempt to apply the method commercially.

Bates (1) concluded that ammonium carbonate and ammonium bicarbonate are too hazardous to fruit to be used in tissue or in a layer in the packed box. Grasovsky and Shiff (2) stated that ammonium bicarbonate as crystals in the fruit wrappers was effective in inhibiting decay by both Penicillium and Diplodia, but had a tendency to increase "physiological breakdown" of the rind. However, they believed that the beneficial effects of the chemical offset the loss caused by the so-called "Nuksans" rind breakdown.

Recently Hopkins, Loucks, and McCornack (3) reported from preliminary tests that ammonium bicarbonate was effective in reducing decay of oranges in fiberboard cartons. The effectiveness of the chemical was increased when moistened to accelerate dissociation.

Because of some of the above indications it was decided to investigate further the use of ammonia as a possible control for decay by the common fungi. During the degreening operation when oranges and lemons are exposed to ethylene gas, the fruits, not having been through the washing and treating processes, are very prone to decay by Penicillium italicum (blue mold) and P. digitatum (green mold). Protection of the fruit with some volatile fungicide during the required 5- to 10-day period seemed highly desirable. Because of our promising results in the control of decay in cardboard shipping cartons with ammonia-emitting compounds, gaseous ammonia was tried on injured and inoculated lemons and oranges in various containers, including minature degreening rooms.

Methods

The fruit was uniformly scratched on the "shoulder" making a wound 1 mm. deep and 1 cm. long. Into the wounds spores of the two molds were introduced either dry with a brush or in suspension by dipping.

The concentrations of the gases were not maintained continuously, as they were by Tomkins and Trout, but intermittently in quantities to give the concentrations shown in the tabulations. After each injection the gases were dissipated by diffusion and absorption. In the experiments with glass jars and stainless steel tanks no means to effect distribution of the gases, except their own properties of pressure and diffusion, were employed. To effect rapid distribution in the miniature degreening rooms the ammonia and ethylene were introduced at the top of the chambers in front of a slowly rotating fan (Fig. 1).

Infection counts were made 1 week after inoculation and the fruits held for observation for 2 additional weeks. The temperature during the period of the experiments was 70° F.

In Table 1 it is seen that 2500 or 5000 ppm of ammonia introduced twice daily for 3 or more consecutive days provided excellent protection of the fruits without causing any apparent injury or off-flavor. Moreover, there was no effect by the ammonia on the acceleration of the rate of degreening by ethylene. A concentration of 10,000 ppm of ammonia caused slight pitting in some experiments and also caused some of the buttons to darken and abscise. The higher concentrations of 20,000 and 50,000 ppm caused severe pitting and darkening of the rind.

Spore viability after 7 days (4 days' treatment followed by 3 days' holding) was determined by scraping off some of the inoculum, mounting it in 5 percent orange juice, and observing the spores for germination. While most of them had been killed a small percentage was still viable. Apparently those in the center of the spore masses were not reached by the fungicide. However, these escapes were ineffective in initiating decay in the treated fruit during the subsequent 2-week holding period.

Table 1. Ammonia gas to protect citrus fruit from blue-green mold decay. Effect of concentration of gas and frequency and number of exposures to the gas.

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Table 1. Ammonia gas to protect citrus fruit from blue-green mold decay. Effect of concentration of gas and frequency and number of exposures to the gas.

*PPM ammonia gas	Type of containers (not sealed)	Date	Ethylene at 500 PPM	Number injections per day	Total number of injections	Decay 7 days after inoculation** Infected fruit Ratio, total fruit of exp.		No. fruit infected, in %		Off-flavor or Injury
						Navel oranges	Eureka lemons	Navel oranges	Eureka lemons	
0, check	jar	11/2/54	No	-	-	45/50	-	90.0	-	
0, "	jar	11/15/54	no	-	-	49/50	-	98.0	-	
0, "	steel tank	11/22/54	no	-	-	104/105	49/76	99.1	64.5	
0, "	jar	11/30/54	no	-	-	50/50	-	100.0	-	
0, "	wood chamber	12/7/54	no	-	-	129/147	138/257	87.8	53.7	
0, "	jar	12/20/54	no	-	-	50/50	-	100.0	-	
0, "	wood chamber	12/28/54	no	-	-	377/385	-	97.9	-	
1000	jar	11/2/54	yes	3	14	19/50	-	38.0	-	none
1000	steel tank	11/22/54	no	3	13	72/213	8/171	33.8	4.7	"
2500	jar	11/30/54	no	2	8	4/50	-	8.0	-	"
2500	jar	11/30/54	no	3	11	2/50	-	4.0	-	"
2500	wood chamber	12/7/54	yes	2	8	2/177	1/266	1.1	0.4	"
5000	jar	11/2/54	yes	3	14	1/50	-	2.0	-	"
5000	jar	11/15/54	no	3	14	1/50	-	2.0	-	"
5000	jar	11/30/54	no	1	4	11/50	-	22.0	-	"
5000	jar	11/30/54	no	2	8	1/50	-	2.0	-	"
5000	wood chamber	12/7/54	yes	2	8	0/171	0/270	0.0	0.0	"
5000	jar	12/20/54	no	2	2	50/50	-	100.0	-	"
5000	jar	12/20/54	no	2	4	42/50	-	84.0	-	"
5000	jar	12/20/54	no	2	6	19/50	-	38.0	-	"
5000	jar	12/20/54	no	2	8	9/50	-	18.0	-	"
5000	wood chamber	12/28/54	no	2	4	54/447	-	12.1	-	"
5000	wood chamber	12/28/54	no	2	6	8/472	-	1.7	-	"
5000	wood chamber	12/28/54	no	2	8	18/472	-	3.8	-	"
10,000	jar	11/2/54	yes	3	14	0/50	-	0.0	-	"
10,000	steel tank	11/22/54	no	3	13	0/199	0/170	0.0	0.0	slight pitting
10,000	jar	11/30/54	no	1	4	2/50	-	4.0	-	none
10,000	wood chamber	12/7/54	yes	2	8	0/180	0/265	0.0	0.0	buttons brown & loose, no pitting
20,000	jar	11/15/54	no	3	14	0/50	-	0.0	-	pitting, darkening around wounds
50,000	jar	11/15/54	no	3	14	0/50	-	0.0	-	very severe injury

* Parts of ammonia to total volume of container.
** Temperature of experiment 70°F.

Table 2. Ammonia gas to protect citrus fruit from blue-green mold decay. Effect of delay of treatment of injured-inoculated fruit using 5,000 ppm ammonia injected twice daily for 3 days.

Type of container (not sealed)	Date	Incubation period (delay) before treatment Hours	Ratio, Infected fruit / Total fruit of exp.		Decay, 7 days after inoculation*. Number of fruit infected, in %	
			Navel oranges	Eureka lemons	Navel oranges	Eureka lemons
Jar	12/28/54	0	0/50	-	0.0	-
"	"	8	0/50	-	0.0	-
"	"	24	2/50	-	4.0	-
"	"	48	38/50	-	76.0	-
"	"	check	42/50	-	84.0	-
Wood chamber	1/4/54	8	38/200	5/215	19.0	2.3
" "	"	24	40/200	9/174	20.0	5.2
" "	"	48	204/211	117/200	96.7	58.5
" "	"	check	180/180	174/177	100.0	98.3
Jar	1/4/54	15	3/50	-	6.0	-
"	"	19	7/50	-	14.0	-
"	"	24	11/50	-	22.0	-
"	"	29	20/50	-	40.0	-
"	"	39	44/50	-	88.0	-
"	"	43	49/50	-	98.0	-
"	"	check	50/50	-	100.0	-

* Temperature of experiment 70°F.

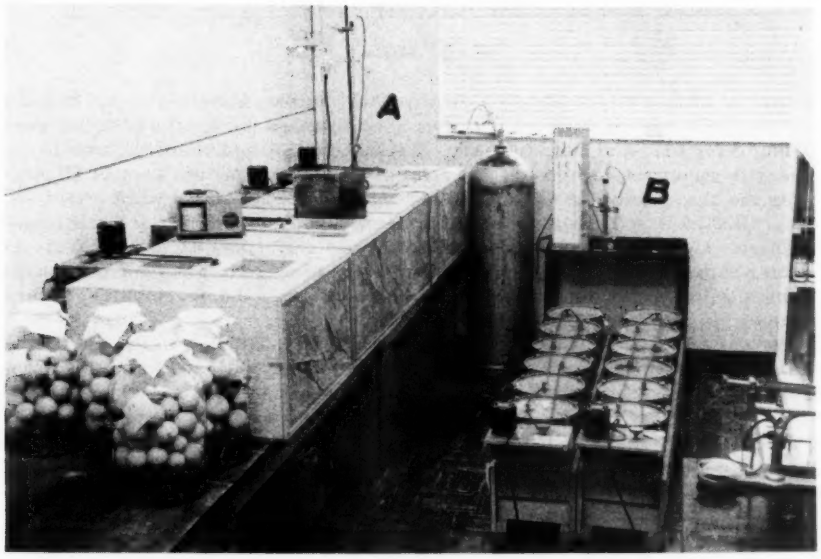

FIGURE 1. Apparatus showing jars in foreground and miniature wooden ethylene chambers in background. Devices for injection of ammonia are shown. A, Direct displacement. B, Calibrated manometer flow.

The data of Table 2 indicate that a treatment of 5000 ppm twice daily for 3 consecutive days will give adequate protection even though inoculation had occurred 24 to 30 hours previous to the first injection of ammonia, and an incubation temperature (70° F.) favorable for decay had been maintained during that period.

The effectiveness of gaseous ammonia when injected into sealed cartons of oranges and lemons is now being determined. The possibility of its use in lemon is also considered.

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FIRE BLIGHT DEVELOPMENT ON APPLE IN WESTERN NEW YORK

W. D. Mills

Striking control of fire blight (Erwinia amylovora) blossom infection on apple and pear by the use of streptomycin sprays during bloom has been obtained during the past two years in California, Ohio, Virginia, and Pennsylvania. Partial control and some failures to control blight on apple with streptomycin sprays has been the experience of workers in Michigan and in New York using the same material and the same timing of sprays which were effective in the warmer areas. Of four New York tests of Agrimycin on pears only one, in Chautauqua County, was severely blighted. In this orchard control by the antibiotic was excellent. This experiment and two tests on King apples where streptomycin greatly reduced blight gave some hope that the antibiotic might be effective in New York if the factors favoring the development of blight were better understood.

A study therefore was made of the development of blight on apples in the Lake Ontario fruit counties since 1917. A compilation of the maximum daily temperatures and precipitation for a 20-day period before the first recorded appearance of blight showed no correlation with its severity. Then the blooming periods of apples from the first bloom of the earliest varieties to the last bloom of late varieties was compiled, together with the maximum daily temperatures and precipitation for these blooming periods and for 30 days after bloom. The information was obtained from the climatological data of the Rochester office of the U. S. Weather Bureau. The severity of blight was compiled using the Plant Disease Survey records for the counties bordering Lake Ontario. Very slight was given a numerical rating of 1, slight 2, moderate 3, severe 4, and very severe 5. The average of the county figures was used as the index of blight severity It was evident that severe blight occurred in years in which maximum daily temperatures of 75° F or higher occurred with precipitation during bloom.

All periods of over 70° F with a trace or more of precipitation at time of bloom were found to be correlated with the incubation time; that is, the greater the number of degree days[1] with precipitation, using 70° F as the base, the shorter was the period from possible inoculation to the first appearance of infections. The correlation coefficient -0.510 was highly significant for 38 degrees of freedom and the regression coefficient was -1.15. In other words, for each increase of 1 degree in maximum temperature for the wet period 1.15 fewer days were required for the development of visible lesions. When the same procedure was followed, using the same temperature base (70° F), but including all periods, with and without precipitation, there was no significant correlation. This suggests that most, and perhaps all, of the infection periods involved both high temperatures and a trace or more of precipitation.

The relationship of temperature and precipitation in bloom with severity of blight was also studied.

The correlation of total degree days plus precipitation in bloom and severity of blight was tested, using various temperatures as well as the conventional 60° F as the base. The following correlations were found; x being the degree days with precipitation in bloom and y the severity index. For the last four correlations only the portion of the degree days between the indicated temperatures is considered, that part above the stated temperature range being excluded from the correlation.

Over	80° F + 0.325	0.05 level
	75° F + 0.479	0.01 level
	70° F + 0.525	0.01 level
	65° F + 0.511	0.01 level
	60° F + 0.474	0.01 level
From	76-80° F + 0.532	0.01 level
	71-75° F + 0.516	0.01 level
	66-70° F + 0.412	0.05 level
	61-65° F + 0.028	not significant

The correlation between the degree days plus precipitation during bloom and the severity of blight is shown graphically in Figure 1.

Correlations were also computed for the degree days in bloom with or without precipita-

[1] The term degree days as used by the United States Weather Bureau is the sum of degrees above 60° F occurring in a given period.

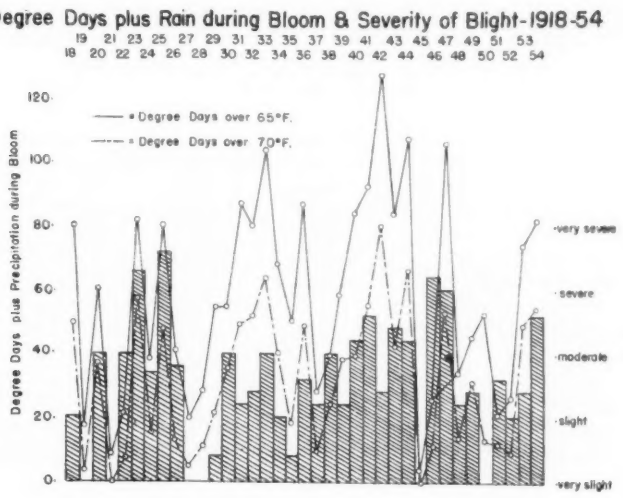
Degree Days plus Rain during Bloom & Severity of Blight-1918-54
19 21 23 25 27 29 31 33 35 37 39 41 43 45 47 49 51 53
18 20 22 24 26 28 30 32 34 36 38 40 42 44 46 48 50 52 54
120
100
80
60
40
20
0
Degree Days plus Precipitation during Bloom
= Degree Days over 65°F.
= Degree Days over 70°F.
very severe
severe
moderate
slight
very slight

FIGURE 1.

tion and the severity of blight.

Over	80^{o} F + 0.633	.01 level
	75^{o} F + 0.308	n.s.
	70^{o} F + 0.276	n.s.
	65^{o} F + 0.351	n.s.
	60^{o} F + 0.330	n.s.

These data show a significant correlation between degree days $\pm$ precipitation only at temperatures over 80^{o}F.

Possible effects of degree days with precipitation during the periods 1-10 days, 11-20 days, and 21-30 days after bloom were tested. None of these 10-day periods were significantly correlated with the severity of blight.

A multiple correlation was also computed using the degree days with precipitation in bloom and the degree days for 1-10, 1-20, and 1-30 days after bloom together with the severity of blight. The effect of the bloom period independent of the period following bloom was thus determined, and the effects of the 10-, 20-, and 30-day periods after bloom independent of the bloom period on severity of blight were computed.

The 10-day and 20-day periods after bloom showed a significant correlation between the degree days over 80^{o} F plus precipitation and the severity. There were no significant correlations below the 80^{o} F level for the 10- and 20-day periods and no signficant correlations at any temperature for the 30-day period.

These data indicate that blossom blight of apple only occurs with maximum daily temperatures during bloom over 65^{o} F, with precipitation or very high humidity.

It is suggested, strictly on a limited trial basis, that the first streptomycin spray should be applied the first day after the beginning of bloom when maximum temperatures exceed 65^{o} F and precipitation or high humidity is forecast. If these conditions do not arrive before full bloom it is suggested the streptomycin should be applied and should give protection up to 7 days if no rain intervenes. If the first spray is applied early in bloom further applications 4 to 7 days later are indicated, being guided by the development of the blossoms and the temperature. If rain follows a streptomycin spray the treatment should be repeated after the rain as soon as temperatures over 65^{o} F with precipitation or high humidity occur. If these conditions do not arrive during the remainder of the bloom period it will be assumed from the above data that further sprays are not needed.

It is not suggested that the successful present schedules of other States should be modified. In the cooler fruit growing areas of western New York some modification of the schedule seems desirable, since the 1954 schedule was not fully effective in this area.

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CONTROL OF FIRE BLIGHT ON FORELLE PEARS WITH ANTIBIOTICS AT HOOD RIVER, OREGON

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The application of four to eight copper sprays or dusts, when started during the early blossom period, has given fair control of pear blight (Erwinia amylovora) in California[2]. A similar program has been in use for some varieties at Medford, Oregon[3]. The fruit is often badly russeted by these copper treatments, but other fungicides tested have not been satisfactory in reducing blight infections. The costly program of cutting out all infected parts, and sterilizing the cut surfaces and pruning tools, is still a standard practice to hold blight in check.

It has recently been found that streptomycin is effective in controlling blight in Bartlett orchards in California[4]. Where blight is an annual problem or where excessive fruit russet occurs from the use of copper fungicides, the use of antibiotic sprays for blight control may prove commercially feasible.

The test in California on Bartlett pears involved the use of only one antibiotic formulation at different concentrations and at various time intervals. The lowest antibiotic concentration that gave control comparable with the copper sprays on Bartlett pears was 30 ppm in five applications. Another test at Hood River, Oregon included four different antibiotic formulations on the highly blight susceptible Forelle pear.

The Hood River Test

In this test 60 trees of the Forelle pear variety were used. The plots were arranged in six replicates of paired trees, which received the following treatments:

1. Check plots received no blight sprays.
2. Zineb (2 lb. to 100 gals. water) (Furnished by E. I. du Pont Co.)
3. A mixture of streptomycin at 100 ppm and Terramycin at 10 ppm (Supplied by the Chas. Pfizer Co., under its brand name Agri-mycin 100).
4. A soluble form of streptomycin sulfate at 100 ppm (Merck and Company's streptomycin sulfate STS).
5. Streptomycin nitrate at 100 ppm (Furnished by Mathieson Chemical Corporation as chemical 1691).

The orchard owner was also furnished enough material of a relatively insoluble form of streptomycin sulfate (Merck's STA) to spray 70 trees four times with a speed sprayer, as a semi-commercial test.

The test plots were sprayed five times. The first application was made April 23 when the trees were in about 80 percent full bloom, and continued at approximately 10-day intervals until June 11. A hand gun was used at a pump pressure of 500 p.s.i. from a small portable spray rig.

In contrast to Bartlett pears, in which most of the infections occur through blossoms, infections commonly occur in Forelle directly through young leaves and tender twig tips. The infections on Forelle usually do not "heal in" where they join lateral branches, which is common in Bartlett, and unless infections are promptly and carefully eliminated the whole tree may become involved.

The first new blight infection was discovered in the test plots on May 25. New infections were cut out of the trees 12 times between June 2 and September 14. The degree of control obtained by the various spray treatments is shown in Figure 1.

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[1] Pathologist, Fruit and Nut Crops Section, Horticultural Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture, Hood River, Oregon.

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PEAR BLIGHT EXPERIMENT--HOOD RIVER, OREGON.
-1954-
FORELLE VARIETY.

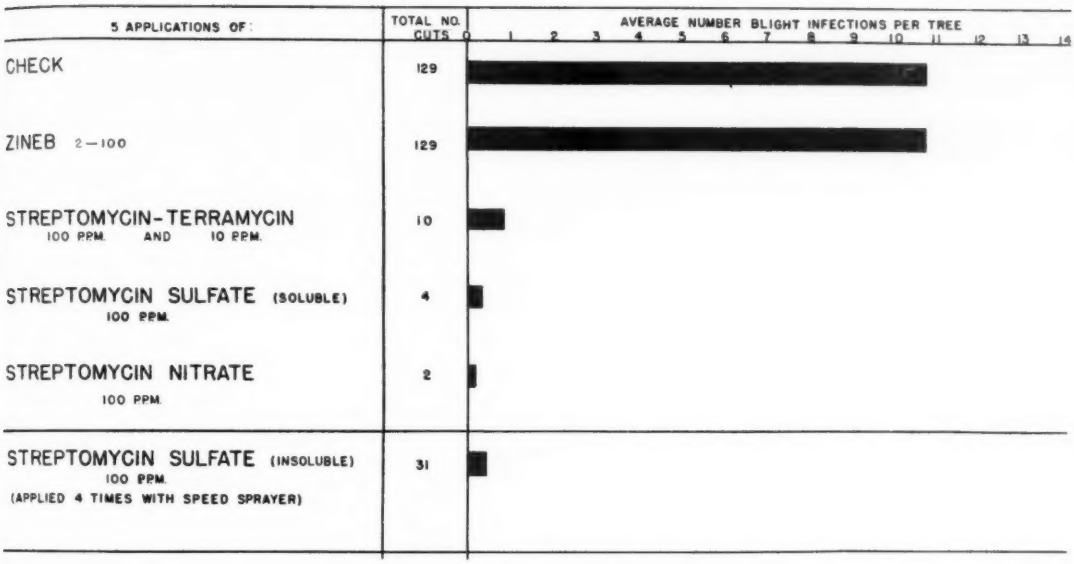

FIGURE 1. Blight infections on Forelle pear trees receiving various spray treatments, Hood River, Oregon, 1954.

Discussion

These tests indicate that antibiotic sprays are highly effective in controlling blight on the extremely blight-susceptible Forelle variety. No fruit injury and only traces of a leaf chlorosis were produced on Forelle pear trees by the antibiotics, whereas copper sprays have previously caused severe fruit russet on this variety. The four different formulations of streptomycin performed equally well when used at the 100 p.p.m. rate.

It has been difficult to raise Forelle pears in many districts because of their extreme susceptibility to blight. Since Forelle fruit often returns a high profit, antibiotics may offer a practical means of blight control for the variety.

HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE, HOOD RIVER, OREGON

THE ANTIBIOTIC CANDICIDIN, A PROTECTANT DIP AGAINST BROWN ROT INFECTION OF PEACH FRUIT

Stanley M. Alcorn and Peter A. Ark[1]

Summary

Of the peach fruits dipped for 1/2 hour in 1:16,666 candicidin and subsequently stored at 32° F for 3 and 21 days before inoculation, 0 and 16 percent respectively became infected, as compared with 22 percent (stored for 3 days only) for the 1:33,333 candicidin- and 66 percent and 100 percent for the 1:5000 captan-treated fruits. None of the fruits treated with 1:1000 liquid lime sulfur were protected. A 5-minute 1:33,333 candicidin treatment was superior to captan and liquid lime sulfur treatments when fruits were inoculated shortly after treatment and after 3 days storage at 32° F. When stored for 3 days at room temperature after treatment and before inoculation, 25 percent of 1:16,666 candicidin-treated fruits became infected (in four trials) as compared with 0 percent (one trial) and 88 percent (three trials) for those treated respectively with 1:5000 captan and 1:000 liquid lime sulfur. However, under the conditions of all of these tests serious fruit injury was associated with captan treatments.

Candicidin solution (1:16,666) stored for 1 week at room temperature was effective in protecting against brown rot infection of fruits dipped for 1/2 hour and stored at 32° F for 1 week before inoculation. Eleven of 12 fruits remained healthy when inoculated shortly after a 1/2-hour dip treatment in the candicidin (1:16,666) stored for 2 weeks.

In vitro experiments have shown that a semi-crude preparation of the antibiotic candicidin[2], at the concentration of 30 parts per million (ppm)[3], will greatly reduce the germination of spores of Monilinia fructicola (1). Initial tests have shown that candicidin does not stop the development of macroscopic brown rot infections, though the rate of development may be reduced. Other experiments, however, have shown that candicidin does have a protective effect against the disease. The results of these tests are reported here.

Except for two trials on the stability of candicidin in suspension, fresh tap water suspensions of approximately 1:16,666 and 1:33,333 (based on the activity of the candicidin, or 1:5000 and 1:10,000 of the semi-crude material per se) were newly prepared for each treatment. Comparison tests were also made with 1:5000 captan[4] and 1:1000 liquid lime sulfur. Tap water was used for the check treatment. To facilitate wetting, Triton X-100[5] was added to all preparations at the rate of 1 ml. per gallon. Peach fruits chosen for testing were without visible blemish near the blossom end and ranged from green to light straw color on the suture. Fruits of the same variety were used for a given test. Fruits of the clingstone varieties Gaume and Stuart, and the freestone varieties Levi, Miller's Late, Salway, and Fossetti were used.

Fruits were picked in the field and were treated either within a few hours of harvesting or after storage at 32° F (2). Treatment consisted of dipping the fruits, usually three per test, in the respective preparations for varying time intervals. Treated fruits were then placed, stem end down, on watch glasses in pyrex containers in such a manner that fruits did not touch each other. The containers were left uncovered for 1 to 3 hours at room temperature (approximately 72° F) to facilitate drying of the fruits. The dishes were then covered and stored at room temperature or at 32° F until time of inoculation. Fruits were inoculated by applying one to two drops of a heavy spore suspension from a 4- to 7-day-old culture of Monilinia fructicola to one of the depressions found near the blossom end of the fruit. Care was taken not to touch the fruit with the dispensing dropper. After inoculation, a little water was added to the dishes and these were then covered and stored at room temperature. Readings were made 7 days after inoculation.

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[1] Respectively, Merck and Co. Postdoctoral Fellow in Plant Pathology, and Professor of Plant Pathology, Department of Plant Pathology, University of California, Berkeley.

[2] Preparation 54R2385 (approximately 30% active) from Merck and Co., courtesy of Dr. John D. Garber.

[3] At the time of our first publication (1) the activity of the candicidin preparation was not known. Therefore, dilutions cited in that paper should be multiplied by the factor 3.3, i.e., 1:500=16,666; 1:10,000=1:33,333, etc.

[4] Technical grade (fines).

[5] Röhm and Haas Co., Philadelphia 5, Pennsylvania.

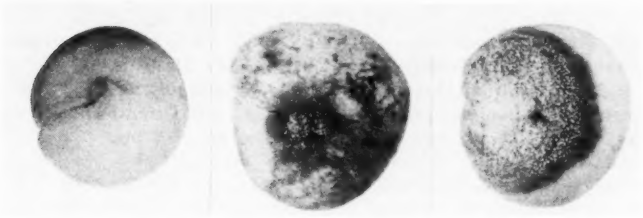

FIGURE 1. Comparative effect of candicidin and captan as protectants against infection of peaches by Monilinia fructicola. Fruits were treated by dipping for 1/2 hour, dried for approximately 2 hours at room temperature, and stored in covered dishes at 32° F for 2 weeks before inoculation. After inoculation fruits were stored at room temperature in moist chambers for one week, when picture was taken. Left, 1:16,666 candicidin, no infection; center, 1:5000 captan, infected (note chemical injury); right, check, infected.

Experimental Results

Under the conditions of these experiments, 94 of a total of 103 fruits dipped for 5, 15, or 30 minutes in 1:16,666 candicidin and stored dry at 32° F up to 3 weeks before inoculation with M. fructicola remained healthy (Fig. 1). Of these, in five experiments none of the 15 fruits dipped for 5 minutes and inoculated within 2 hours became infected. Two of 17 fruits in six tests dipped for 30 minutes and similarly treated became infected. Single experiments showed that of fruits dipped for 5 minutes, 2 of 3, 1 of 3, 0 of 3, and 2 of 3, became diseased when stored at 32° F for 3, 7, 14, and 21 days respectively before inoculation. In tests replicated two or three times, 0 of 6, 0 of 9, 0 of 6, and 1 of 6 fruits dipped for 30 minutes became diseased when inoculated after comparable storage periods. Of 18 fruits dipped in 1:33,333 candicidin for 5, 15, and 30 minutes, in duplicated experiments, and inoculated within a few hours of treatments, only 1 became infected. Of 27 fruits similarly treated in tests replicated three times, but stored 3 days at 32° F before inoculation, 8 became infected. The percentage of fruits showing disease did not differ appreciably between the lots treated for 5 and for 30 minutes. In experiments comparable to those with candicidin, 57 of 57 fruits treated with 1:1000 liquid lime sulfur became infected, as did 38 of 48 fruits dipped in 1:5000 captan. Of these, infected fruits ranged from 66 to 100 percent in the tests involving the various treatment and storage periods. Of 114 fruits dipped in water and used as checks in 38 tests 113 became infected when inoculated after the appropriate storage period.

Several other tests were made to determine the effect of storage for 3 days at room temperature, before inoculating with M. fructicola, on the efficacy of 1/2-hour dip treatments. Three of a total of 12 fruits treated with 1:16,666 candicidin, in four trials, became infected with brown rot as compared with the absence of infections in 3 fruits treated in one trial with 1:5000 captan and 8 infections of 9 fruits in three trials with 1:1000 liquid lime sulfur.

All captan-treated fruits showed discoloration from chemical injury (Fig. 1). The severity apparently varied with the length of treatment time and ripeness of fruit at time of treatment. No injury was noted on fruits treated with candicidin or liquid lime sulfur.

To determine the stability of liquid candicidin in suspension, 6 peach fruits in each of 2 tests were dipped for 1/2 hour in a 1:16,666 candicidin (plus 1 ml. per gallon of Triton X-100) suspension which had stood for 2 wekks at room temperature. The fruits were then air dried and inoculated with M. fructicola. Eleven of the 12 fruits remained healthy while all checks were infected. Three fruits dipped for 1/2 hour in 1-week-old candicidin suspension and then stored dry for 7 days before inoculation also remained free of infection.

It appears that candicidin has no protective effect against Mucor sp., which was present in some experimental chambers as a contaminant.

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EFFECTIVENESS OF STREPTOMYCIN AS A CONTROL FOR COMMON BACTERIAL BLIGHT OF PINTO BEAN[1]

Robert B. Marlatt[2]

Summary

Pinto bean plants were sprayed with aqueous solutions of streptomycin sulfate at strengths of 240 ppm and 1000 ppm, and Agrimycin 240 ppm and 1000 ppm, in an attempt to control common bacterial blight caused by Xanthomonas phaseoli (E. F. Sm.) Dowson. In addition, plants were dusted with 240 ppm streptomycin sulfate. Four applications of these materials failed to give significant control of the disease.

Infected pinto bean seeds were soaked for 15 minutes in a 1:500 aqueous dilution of streptomycin sulfate and planted in non-infested soil. The treated seed produced plants with as high an incidence of infection as in the controls.

Streptomycin has been reported as a control for halo blight of beans (1, 2, 3, 4, 5) and for common blight (1, 3). Epidemics of common bacterial blight of pinto beans occur annually in Arizona's Yavapai County; therefore, the possibility of using streptomycin as a means of control was investigated.

Methods

Sprays and Dust: Antibiotic sprays and dust were applied to plots in a commercial field of pinto beans situated near Chino Valley, Arizona. The beans were planted on May 25, 1954 and four applications were made on June 11, July 2, July 23, and July 30.

The materials applied included an aqueous solution of streptomycin sulfate (Merck & Co., Inc.) plus a wetting agent, at strengths of 240 ppm and 1000 ppm, Agrimycin (Chas. Pfizer & Co., Inc.) in amounts to provide 240 ppm and 1000 ppm of streptomycin, and a dust formulated to contain 240 ppm streptomycin (White Chemical Co.). Control plots received no dusts or sprays.

Two experiments were conducted simultaneously in the same field, each one consisting of a 6 x 6 Latin square. Sprays were applied by means of a hand sprayer with three cone nozzles at a rate exceeding 100 gallons per acre; the dust was applied at a rate exceeding 200 lbs. per acre by the use of hand-operated rotary duster.

Seed Treatment: Smith (4) reported control of all but a trace of seed-borne infection by halo blight of Sensation Refugee beans by soaking the seeds for 15 minutes in a 1:500 dilution of streptomycin sulfate in water.

Pinto bean seeds produced in Arizona in a field that showed severe damage by common blight were soaked in an aqueous solution of streptomycin sulfate at a dilution of 1:500 for 15 minutes, dried, and planted in a field that had never contained beans. Soaked and untreated beans were planted in plots each of which consisted of eight adjacent rows 20 feet long and which were replicated four times.

Results

Sprays and Dust: At the time that the third application was made, July 23, common blight was becoming severe on all of the plants, treated and untreated alike. Shortly before harvest no differences could be detected between any of the treatments; the entire field was severely damaged by the disease.

Seed Treatment: The numbers of diseased plants in both the seed-treatment and control plots remained high throughout the season. The earlier counts were taken before any secondary spread of the disease was likely to have taken place; no rains had occurred and no equipment had

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[1] Arizona Agricultural Experiment Station Technical Paper No. 352.

[2] Assistant Plant Pathologist, University of Arizona Agricultural Experiment Station, Mesa, Arizona.

been used in the field.

Discussion

In an area such as Chino Valley, where common bacterial blight is so severe, four applications of streptomycin and Agrimycin, at strengths as great as 1000 ppm, fail to give any evidence of control. The costs of more frequent applications would be prohibitive even if a greater number of sprays showed promise of controlling the disease.

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5. Zaumeyer, W. J., H. R. Thomas, J. W. Mitchell, and H. H. Fisher. 1953. Field control of halo blight of beans with streptomycin. Phytopath. 43: 407

ARIZONA AGRICULTURAL EXPERIMENT STATION, MESA

PROGRESS REPORT ON THE USE OF STREPTOMYCIN FOR THE CONTROL OF BACTERIAL SPOT OF TOMATOES UNDER FIELD CONDITIONS ON SOUTH FLORIDA SANDY SOILS

Donald M. Coe

Since the publication by Conover[1] of his results on the control with streptomycin of bacterial spot (Xanthomonas vesicatoria (Doidge) Dows.) of tomatoes in seed beds in Southern Florida, there has been great interest in the use of this material in other tomato-growing areas. The growing practices followed and the climatic factors involved in any one tomato-growing area in Florida may be so different from those of another area that the results obtained in one locality may find only limited application in another. This is particularly true of the mature-green tomato industry. For instance, the tomato industry in the Homestead area is primarily based on a seed bed and transplant system on marl soils. On the sandy soils of the Fort Pierce area, approximately 160 miles north of Homestead, the industry is based on a direct seeding system. Seeding for the fall crop is carried out during the period from August 15 to September 10 which is 3 to 4 weeks earlier than in the Homestead area. This subjects the plants to a longer exposure to the summer rainy season, which is an important factor in the active and destructive spread of this disease. The work reports preliminary results of field trials with streptomycin under conditions of rainfall and winds favorable to spread of the disease.

Materials and Methods

For the evaluation of streptomycin, a commercial preparation[2] was used in a set of variety trials in which a heavy infection of bacterial spot was already established at the time of first application. Sixteen varieties replicated four times in 17-plant plots (spaced 2 feet apart) were bench-seeded on August 6 and transplanted to the field on August 27 on raised beds. At the time of transplanting there was no evidence of any bacterial spot infection in any of the plants. It was first observed in the plots on September 20 when the plants were 12 to 14 inches high, and the first hand was in bloom. Within a few days the infection was general.

The four blocks of the experiment were designated as A, B, C, and D, and the streptomycin was applied to blocks A and C on the schedule outlined below:

September	24,	400 ppm,	Approximately 75 gal. per acre
"	28,	"	"
October	4,	"	100
"	8,	"	"
"	11,	"	150
"	15,	200	150-175
"	20,	"	"
"	26,	"	"

With one exception at least 24 hours elapsed with no rain following the spray applications. The October 8 application was followed by rain within 3 hours and a total of 3 1/2 inches of rain fell within the next 48 hours.

The entire planting (Blocks A, B, C, and D) was sprayed on a 6- to 7-day schedule with zineb, parathion, and DDT. At least 24 hours were always allowed between the applications of streptomycin and the fungicide-insecticide sprays. All applications were at 200 to 300 pounds pressure with nozzles adjusted to direct the spray to the sides as well as the tops of the plants. The number of nozzles ranged from three per row in the earlier applications to nine in the final applications.

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[1] Conover, Robert A. 1954. Control of bacterial spot of tomato and pepper seedlings with Agrimycin. Plant Dis. Reptr. 38: 405-409.

[2] Agrimycin 100, a trademarked formulation prepared and furnished by Chas. Pfizer and Co., containing 15% streptomycin and 1.5% oxytetracycline (Terramycin).

Table 1. Average indices[a] of bacterial leaf spot infection on tomato plants on three dates in streptomycin-treated and non-treated blocks.

Treatment	Disease index		
	Oct. 11	Oct. 19	Oct. 30
Non-treated (B plus D)	6.53	7.14	8.80
Treated (A plus C)	2.86	2.08	2.09

[a] 1 = no disease; 10 = spotting severe and well distributed into the tips of the plant, defoliation in excess of 50 percent.

Table 2. Yields and average fruit size from streptomycin treated and non-treated blocks of tomato plants.

Harvest and date	Total number fruit		Total weight fruit (lbs.)		Average size fruit (lbs.)	
	Treated	Non-treated	Treated	Non-treated	Treated	Non-treated
1st (Nov. 1)	1208	1190	397.90	326.80	.329	.275
2nd (Nov. 13 and 15)	4707	3984	1266.30	798.60	.269	.202
3rd (Nov. 23 and 24)	5154	5198	1089.40	842.95	.211	.162
4th (Dec. 7)	4813	3960	863.35	579.35	.179	.146
Totals and averages	15882	14332	3616.95	2547.70	.228	.178

Table 3. Severity of bacterial spot based on spots-per-fruit in streptomycin treated and non-treated tomato plots from first 3 harvests.

Harvest and date	Fruit spotted				Percentage distribution of spots per fruit					
	Number		Percent		1-10		11-25		26-00	
	Treated	Non-treated	Treated	Non-treated	Treated	Non-treated	Treated	Non-treated	Treated	Non-treated
1st (Nov. 1)	416	640	34.44	53.78	76.92	61.88	15.87	21.41	7.21	16.72
2nd (Nov. 13 and 15)	1178	1545	25.03	38.78	93.72	83.43	5.18	13.85	1.10	2.72
3rd (Nov. 23 and 24)	894	2088	17.35	40.17	96.87	90.33	2.35	8.24	0.78	1.44
Total or average	2488	4273	22.48	41.20	92.04	83.57	5.95	12.24	2.01	4.19

Weather Data

Because the occurrence of driving rains is so important in the incidence and spread of bacterial spot, the following narrative of the prevailing weather is presented to be considered in its relation to the arithmetical data. The period covered dates from the time of transplanting (August 27) to final harvest (December 7).

From August 27 to 31 rain occurred on 5 days with a total of 0.63 inches. During September a total of 12.01 inches was recorded at the plots with rain on 22 days. On September 1, 15, 19, and 24 rainfall in excess of 1 inch was recorded. This record shows ample rain in a distribution pattern ideal for the spread and build-up of bacterial spot. In October rain fell on 13 days with a total of 6.98 inches. On October 3, 1.96 inches was recorded; October 10, 2.35 inches; October 11, 0.98 inches; and on October 24, 0.49 inches. Thus the precipitation for the first half of this month was also favorable to disease development and a continuation of the conditions prevailing during September.

No rain fell in November until the 10th, but from then until the 23rd rain was recorded on 10 days for a total of 4.25 inches. A fall of 1.25 inches occurred on November 11 and a heavy, driving rain of 2.40 inches fell on November 15. This last rainfall spread the infection rapidly throughout the plots. The last application of streptomycin had been made on October 26 and all or most of its protective benefits were gone by the time this last rain fell.

Results

Disease Ratings: On October 11, 19, and 30 the plots were rated on a 1 to 10 scale for disease severity, with 1 representing a plant with no disease and 10 a plant with severe spotting well distributed into the tips of the plant as well as defoliation in excess of 50 percent. Other criteria considered in the scoring were vertical distribution of leaf spots in the plant, yellowing of affected leaves and "firing" of the lower leaves.

Although some variation occurred throughout the plots in the scores assigned, the general picture was one of uniformity. In the first rating on October 11 there were still some differences due to an infection gradient throughout the plots. This was progressively less evident as time passed until the highest degree of uniformity was shown in the October 30 readings. Some differences in varietal response were evident but substantive observations must be made before any conclusions can be made in this matter. A summation of the ratings is shown in Table 1.

Two conclusions are made from the rating data. 1 -- In the non-treated plots the disease became more serious as time passed. In early November (several days after the last reading) the entire area of the non-treated plots would have been scored at 10. 2 -- In the treated plots there is shown a degree of recovery. This is due to the production of spot-free growing tips and the absence or reduction of subsequent spread after the first waves of initial infections. The treated plots became stabilized at a low level of disease development and remained so until the the termination of the experiment.

Yield and Harvest Data: Four harvests of the fruit were made and data recorded on yield and size of fruit (Table 2). The effect of the disease in the absence of streptomycin applications is evident in a reduction in yield, a reduction in size of fruit, an increase in percentage of spotted fruits and an increase in severity of spotting per affected fruit. An increase in yield of 1069.25 pounds of fruit was recorded, which is an increase over no treatment of 41.97 percent. In the size of fruit an increase of 0.05 pounds per average fruit is an increase of 28.09 percent. A total increase of 1550 fruits is an increase of 10.81 percent.

On the basis of the first three harvests the treated plots, as shown in Table 3, showed a decrease in spotted fruits of 18.72 percent. While this may appear to be a considerable decrease, a total of 22.48 percent of the fruits spotted in the treated plots cannot be considered as a satisfactory achievement. The number of spots per fruit was somewhat higher in the non-treated plots than in the treated plots. However, the gradient as scored on a 1-10, 11-25, and 26-indefinite rating was considerably greater in the first harvest and became less as the season progressed. On an average between 83 and 92 percent of all spotted fruits in both treatments fell into the 1-10 spots per fruit class, but the greatest differential is found in the 26-indefinite class.

Supplemental Observations

In addition to the plots on which quantitative data were taken, observations were made on

adjoining tomato plantings which were subjected to the same infection and disease development potential. The spray programs were somewhat different but all had the same basic zineb-parathion-DDT schedule as stated above in addition to their respective streptomycin applications. Immediately adjoining the trial just reported were 12 rows containing 48 non-replicated variety plots of the same size, arrangement, and history as the plots from which the above data were taken. All 12 rows were sprayed with streptomycin at the same times and rates; i. e., five applications at 400 ppm and three applications at 200 ppm. There were no intervening, non-treated blocks to serve as possible added sources of inoculum. Since each of the 48 plots in this planting was a distinct varietal entity with no paired plot for comparison, it appeared advantageous to treat it throughout and observe the overall effects of an intensive streptomycin program. It may be stated that under these conditions the control appeared to be better than in the treated blocks from which the harvest and disease severity data were recorded.

Adjoining the area above was a planting of 12 rows of tomatoes that were direct-seeded on September 4 and thinned to 2-foot intervals on September 30. At this time they were in the second or third true-leaf stage and bacterial spot had been observed generally distributed throughout the planting on September 20.

The planting was divided longitudinally into 4 blocks of 3 rows each. Alternate blocks were sprayed with streptomycin as outlined previously except that five applications were made at 200 ppm and three applications made at 100 ppm. In keeping with the size of the plants sufficient gallonage was used to give adequate coverage. The total number of gallons actually applied to the plants was less than in the other trials and considerable material was dispensed directly to the soil.

Several observations during the progress of the experiment failed to disclose any build-up of the infection. With the dropping of the infected basal leaves the plants appeared to be clean in all blocks except for only occasional spots when spraying was terminated on October 26. However, enough inoculum was present to provide for a severe spread under the influence of the heavy rains of November 11 and 15. The protective potential of the eight applications had been exhausted between October 26 and these dates of heavy rainfall.

Discussion

A considerable degree of success in the control of bacterial spot of tomatoes through the use of streptomycin is recorded. This has been measured by visual ratings of disease severity on the vines, by fruit harvest data, and by the severity of fruit spotting. Certain weather data are presented to show the correlation of climatic factors with incidence and spread of the disease throughout the plots. The data show that a high degree of control of the foliage phases of bacterial spot was accomplished with the methods employed. This is reflected in an increase in yield and in average size of fruit. A decrease in the incidence of fruit spotting was obtained but it is less than can be considered good commercial control. In connection with these data must be considered the program of five applications of streptomycin at 400 ppm followed by three applications at 200 ppm. Approximately 500 gallons of the 400 ppm solution were used per acre followed by approximately 450 gallons of the 200 ppm solution. Although the work reported herein shows progress in the control of bacterial spot on tomatoes, it furthermore shows that much more data must be obtained on the timing of the applications in relation to rainfall and plant development, as well as on rates and methods of applications. The conclusion must also be made that the results of Conover with the same disease on tomatoes but in another area, and the work of Cox et al[3]. with the same disease on peppers, cannot be interpolated in their entirety toward control of bacterial spot under a different set of growing conditions. Much additional work is to be done before local recommendations can be made for the use of streptomycin as a general control material for bacterial spot.

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[3] Cox, R. S., W. R. Comegys and J. W. Heuberger. Preliminary tests with antibiotics for the control of bacterial leafspot of pepper. Trans. Peninsula Hort. Soc. 1953.

A NEW ANTIFUNGAL AGENT, FILIPIN.

David Gottlieb, Alfred Ammann, and H. E. Carter

A hitherto undescribed streptomycete produces a metabolite which is extremely inhibitory to the growth and germination of fungi. It has many properties which might favor its use as a disease control agent of plants.

This metabolite has been isolated and partially purified. In this state the compound, filipin, is a yellow solid that is extremely insoluble in water. Ultra violet light absorption studies revealed three strong absorption peaks which indicate that filipin belongs to the polyene family of antibiotics. It is different from any of the members of this series which have been reported in the literature. Negative results were obtained with the following tests: Molisch, Benedicts, Tollens, ninhydrin, biuret and ferric chloride. Filipin contains no nitrogen, sulfur, or halogen. An approximate analysis of the amorphous material showed C= 61.98% and H= 8.99%.

Filipin is relatively non-toxic to plants. Concentrations of 200 µg per ml alcohol solution did not retard the germination of peas or tomatoes in Petri plates. Experiments in which the antibiotic was applied as a slurry to nine different kinds of seeds showed no important symptoms of toxicity to the plant. In no case was there any significant reduction in stand of the plants. Seven of the crops showed absolutely no discernible difference between the treated and untreated plants. In two cases slight differences could be seen: the emergence of tomatoes were "set back" for one day and the final height of the treated cucumbers was a bit uneven. Pea seeds which were immersed in solutions of the antibiotic then left in petri dishes to germinate were almost completely free of rot due to fungi, whereas untreated seeds contained Aspergilli, Penicillia, and Phycomycetes. Spraying young tomato plants with 830 ppm of the antibiotic prep did not produce any phytotoxic symptoms. The incidence of gray leaf spot of tomatoes was reduced 25 percent under a similar treatment when the plants were artificially infected in the greenhouse. Fifty ppm of this filipin preparation prevented the germination of spores of Stemphylium solani in dilution tests on solutions of the antibiotic.

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CONTROL OF DOWNY MILDEW ON CUCUMBERS AND MUSKMELONS WITH TANK MIXTURES OF A FIXED COPPER AND A DITHIOCARBAMATE

F. S. Beecher

For control of downy mildew, Pseudoperonospora cubensis (Berk. & Curt.) Rostow., preferences for fungicidal sprays have varied in different areas, fixed coppers, zineb, and ziram being most generally used. In trials at Beltsville in 1952, when other diseases were not present to confuse the picture, zineb (Dithane Z-78) gave good control of downy mildew on cucumbers and was superior to Tri-basic copper sulfate. However, Tri-basic copper sulfate very effectively controlled angular leaf spot, Pseudomonas lachrymans (E. F. Sm. & Bryan) Carsner, of cucumber, while Dithane Z-78 was ineffective. Therefore tests were begun in 1953 to determine what a combination of Tri-basic copper sulfate with a dithiocarbamate might do in controlling both downy mildew and angular leaf spot on cucumbers and downy mildew on muskmelons.

In the past, tank mixtures of a fixed copper with a dithiocarbamate have been thought to produce foliage injury, but tests for two years at Beltsville have shown no such injury attributable to the combined spray materials. Plots of 48 plants each, replicated four times, were used. In this location the early summer is usually warm to hot with high humidity, becoming hotter and usually drier as the season advances. Possibly where the weather is cool and rainy the results might be somewhat different.

In the 1953 tests a tank mixture of Tri-basic copper sulfate with Dithane Z-78 (3:2:100) gave the best mildew control on cucumbers, while on muskmelons a Tri-basic copper sulfate-ziram (Zerlate) mixture (3:2:100) appeared somewhat better. This apparent difference may have been due to the slow progress of mildew and the appearance of considerable infection by Alternaria cucumerina (Ell. & Ev.) J. A. Elliott, on the melon foliage, where it may have been controlled better by the Tri-basic copper sulfate-Zerlate mixture. Even for control of angular leaf spot on cucumbers, the Tri-basic copper sulfate-Dithane Z-78 mixture was a close second to the Tri-basic copper sulfate alone. As the mixtures of the fixed copper and dithiocarbamates caused no foliage injury, it seemed desirable to continue the tests, especially as these mixtures seemed to give better downy mildew control than any of the ingredients alone.

In 1954, tank mixtures of Tri-basic copper sulfate with zineb or ziram, as well as alternate sprays of the same materials, were tried again. Field tests on both cucumbers and muskmelons showed that a tank mixture (3:2:100) of Tri-basic copper sulfate with Dithane Z-78 gave better control of downy mildew than any other materials tested. This mixture proved even better than a spray of Tri-basic copper sulfate (4:100) alternated with Dithane Z-78 (2:100).

All sprays were applied at 7-day intervals at pressures of about 200 pounds. A spreader ("Tween 20") and an insecticide ("Tubacide", a rotenone extract) were added to all sprays. No foliage injury due to the spray materials was seen at any time.

These limited tests included only combinations of Tri-basic copper sulfate with various dithiocarbamates, and further testing will be necessary to determine what results might be obtained with other strengths of these materials, or with combinations of other fixed coppers with dithiocarbamates. Up to now, however, the Tri-basic copper sulfate-Dithane Z-78 mixture at 3:2:100 has given the best control of downy mildew. Thus far it appears that certain mixtures of Tri-basic copper sulfate with a dithiocarbamate possess some fungicidial value not inherent in the single ingredients when used alone.

HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, PLANT INDUSTRY STATION, BELTSVILLE, MARYLAND.

PARASITIC OR SUSPECTED PLANT PARASITIC NEMATODES FOUND IN RICE SOILS FROM TEXAS AND LOUISIANA

John G. Atkins, Max J. Fielding, and John P. Hollis

The recent discovery[1] of Radopholus oryzae (van Breda de Haan) Thorne at the Rice-Pasture Experiment Station, Beaumont, Texas in October 1954, prompted a survey of the occurrence of nematodes in the rice area of Texas and Louisiana. A total of 76 samples from rice fields scattered throughout Texas and Louisiana and 25 samples from the Beaumont Station were collected. The samples consisted of soil and rice roots or the basal portions of rice plants with surrounding soil. The rice had already been harvested in most fields when the samples were collected but many of the cut plants had developed new tillers. In the laboratory the nematodes were separated from the plant and soil samples by appropriate methods, utilizing a 325-mesh per inch sieve. These were carefully examined microscopically for possible parasitic nematodes. The nematodes found in the samples that were considered as parasitic or possibly parasitic on rice are listed in Table 1. Large numbers of free-living nematodes (saprophytic or predacious) were also observed.

Table 1. Parasitic or suspected parasitic nematodes found in samples of rice soils from Texas and Louisiana.

Nematode	Number of times found	
	Texas (39 samples)	Louisiana (37 samples)
A. Parasitic Forms		
1. Radopholus oryzae (v. Breda de Haan) Thorne	4	10
2. Ditylenchus sp.[a]	2	0
3. Ditylenchus sp.[a]	1	1
4. Pratylenchus sp.[a]	3	0
5. Tylenchorhynchus sp.[a]	29	21
6. Tylenchorhynchus sp.[a]	2	0
7. Meloidogyne sp.[b]	0	1
8. Helicotylenchus sp.[a]	1	3
9. Hoplolaimus sp.[a]	0	1
10. Rotylenchus sp.[a]	1	0
11. Criconemoides sp.[a]	0	2
B. Suspected Parasitic Forms		
1. Psilenchus hilarulus de Man	7	6
2. Psilenchus aberrans Thorne	1	1
3. Psilenchus sp.[a]	5	8
4. Tylenchus sp.[a]	10	15
5. Dorylaimellus virginianus Cobb	4	3
6. Dorylaimellus sp.[a]	1	5
7. Longidorus sp.[a]	1	2

[a] Undescribed species.

[b] Only males and larvae found in sample.

[1] Atkins, J. G., Max J. Fielding, and John P. Hollis. 1955. A new nematode on rice in Texas and Louisiana. Plant Dis. Reptr. 39: 69.

The nematodes listed as parasitic forms belong to genera containing known plant parasitic species. It should be emphasized that the pathogenicity of the nematodes listed has not been proved except for R. oryzae, females of which were dissected from inside a rice root. Those so listed are believed to be potentially capable of causing damage since they were found in association with rice roots. Although Steiner[2] as well as others reported that several genera of nematodes have been found on rice, a literature review is not included.

It is planned to study certain of these nematodes and to evaluate their economic importance on rice. Pure populations of some have been established in the greenhouse for pathogenicity and other basic studies. Greenhouse or field studies planned include pathogenicity, host range, varietal response, and soil fumigation, as well as the effect of crop rotation, irrigation, and summer fallow practices upon nematode populations. Various root parasitic fungi will also be studied in relation to nematodes as causes of root disorders.

FIELD CROPS RESEARCH BRANCH AND HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE, THE TEXAS AGRICULTURAL EXPERIMENT STATION, AND THE LOUISIANA AGRICULTURAL EXPERIMENT STATION, COOPERATING

[2] Steiner, G. 1934. Root-knot and other nematodes attacking rice and some associated weeds. Phytopath. 24:916-928.

1, 2-DIBROMO-3-CHLOROPROPANE -- A NEW NEMATOCIDE[1]

C. W. McBeth[2] and Glenn B. Bergeson[3]

Abstract

The nematocidal effectiveness of 1, 2-dibromo-3-chloropropane, previously known as Shell OS 1897, has been demonstrated in the greenhouse and field to be 8 to 16 times that of D-D and 2 to 5 times the effectiveness of ethylene dibromide.

Although some crops, such as onion and tobacco, are extremely sensitive to this material, certain established plants such as citrus, walnuts, peach, and fig will tolerate dosages up to 20 times the nematocidal rate without injury.

The importance of C_3 and C_4 unsaturated halides as soil fumigants for the control of nematodes and fungi is well known. Dichloropropene-dichloropropane mixture (1), dichlorobutene (5), dibromobutene (4), chlorobromopropene (3), and allyl bromide (2) are examples. This paper reports some preliminary observations on the nematocidal effectiveness of a saturated chemical, 1, 2-dibromo-3-chloropropane. This material is a heavy, straw colored liquid with a specific gravity of 2.08, weighing 17.3 pounds per gallon. It boils at 196° C and has a vapor pressure of 0.8 mm at 70° F (21° C). Its flash point (tag open cup) is above 180° F.

The nematocidal effectiveness of 1, 2-dibromo-3-chloropropane has been determined in greenhouse and field tests. The technique followed in the greenhouse tests was as follows: gallon crocks were filled with Fresno sandy loam soil known to be heavily infested with rootknot nematodes (Meloidogyne incognita var. acrita). The test chemicals were pipetted into the center of the crock at a depth of 3 inches. Soil moisture in these tests was held at about 75 percent of moisture equivalent. The pots were not watered during the first 48 hours, but were watered lightly thereafter for the duration of the test. After 7 days at 80° F, the soil in the crocks was transferred to eight 4-inch clay pots which were immediately planted with tomato seeds and watered. Approximately 6 weeks later, the soil was washed from the tomato roots and the extent of nematode attack noted. Further details of the technique used in these tests have been published elsewhere (6).

Field tests were made in a variety of ways, but the results reported in Table 1 were obtained from replicated plots 6 feet square, in heavily rootknot infested soil, to which the chemical had been applied with a precision hand-injector at 8-inch dept on 12-inch centers. After a crop of summer squash had been grown, the roots were examined for evidence of rootknot nematode attack.

Table 1. Minimum effective dose of 1, 2-dibromo-3-chloropropane compared with two well-known nematocides.

Material	Greenhouse Ml./gallon crock	Field Gallons/acre
Dichloropropene-dichloropropane mixture	0.8	20
Ethylene dibromide	0.2	5
1, 2-Dibromo-3-chloropropane	0.05-0.1	1.25 - 2.5

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[1] Previously known as Shell Experimental Nematocide OS 1897.

[2] Senior Nematologist, Agricultural Research Division, Shell Development Company, Modesto.

[3] Senior Laboratory Technician, Department of Plant Nematology, University of California, Berkeley, California; formerly Junior Nematologist at the above Shell Laboratory.

From these data, it appears that against rootknot nematode, 1, 2-dibromo-3-chloropropane is from two to five times as effective as ethylene dibromide and eight to sixteen times as effective as D-D[4]. Similar ratios of effectiveness have been observed in greenhouse and field tests against sugar beet nematode (Heterodera schachtii).

Table 2. Preliminary indications of phytotoxicity of 1, 2-Dibromo-3-Chloropropane.

TOLERANT	INTERMEDIATE	SENSITIVE
Established Plants		
Citrus trees	Sweetpotatoes (transplants)	Tobacco transplants
Peach trees	Cole crop transplants	Onion transplants
Fig trees	Grape cuttings	Irish potatoes (seed pieces)
Walnut trees	Tomatoes (seed or established)	
Grape vines		From Seeds
Sugar beets (side dress)		
Hibiscus		Tobacco
Rose		Onion
Strawberry		Cole crops
From Seeds or Bulbs		Garlic
		Lettuce
Carrots		Sugar beets
Beans		Garden beets
Peas		
Squash		
Corn		
Radishes		
Cotton		
Croft lily		
Peanuts		

Most nematocides are quick-killing compared with 1, 2-dibromo-3-chloropropane. Living rootknot nematode larvae can be screened from treated soil several weeks after treatment with this material. These larvae are affected in some way that prevents their infecting plants, since tomatoes grown in such soil will have perfectly clean roots.

Probably the most promising and unique property of 1, 2-dibromo-3-chloropropane is its lack of phytotoxicity on plants with established root systems. Living citrus, peaches, figs, walnuts, grapes, and several ornamentals have been treated with from 2 to 20 times nematocidal doses without injury.

Some row crops such as onions and tobacco are extremely susceptible to the chemical, especially as seedlings germinating in treated soil. Others seem tolerant of nematocidal doses even when seeded or transplanted at the time of treatment.

Experiments involving phytotoxicity have been preliminary. The classification offered in Table 2 may, however, be useful in planning field tests.

Field tests in various parts of the United States on several crops have been conducted during the 1953 and 1954 seasons. Most of these have been cooperative with U. S. Department of Agriculture and State experiment workers. The results of these tests are being reported elsewhere.

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INCIDENCE OF VERTICILLIUM WILT IN COTTON NOT AFFECTED BY ROOT-KNOT NEMATODES

W. D. McClellan[1], Stephen Wilhelm[2], and Alan George[3]

Verticillium wilt (V. albo-atrum) is recognized as one of the major diseases of cotton, and is particularly serious in the lower Mississippi Valley and in the irrigated region of the Southwest (4). It is the principal disease of cotton in the San Joaquin Valley of California. Root-knot nematodes (Meloidogyne incognita var. acrita) are also serious in cotton in California (5). There are numerous reports indicating that attacks by root-knot nematodes (Meloidogyne spp.) or by the sting nematode (Belonolaimus gracilis) increase the severity of Fusarium wilt of cotton (2, 3, 6). There are no such reports indicating that attacks by root-knot nematodes result in an increase in the severity of Verticillium wilt in cotton. An opportunity for information on this point occurred in a field at Woodville, California, in 1953.

This field was known to have a severe root-knot nematode history. Sugar beets grown in 1950 in this field of Foster Fine Sandy Loam had to be disced up and the field replanted to milo because of nematodes. It was planted to cotton in 1951 and 1952 and root-knot nematodes were serious in both years. In 1953 we used ethylene dibromide in replicated test plots in this field[4]. Eighty-five percent ethylene dibromide (EDB)[5] was used at the rate of 4.5 gallons per acre either as a preplanting application or as an application at the time of planting. Applications were made on the flat and not on ridges. The chisels on the applicator were set to apply the EDB at a depth of 8 inches in rows 12 inches apart. The plots were 600 feet long and 15 feet wide. The preplanting application was made on April 13 and the planting application was made on April 22, just ahead of the planter. Five rows of cotton were planted per plot. There were four replications of each treatment and a third plot in each replicate was left untreated.

Excellent nematode control was obtained by either of the EDB treatments, as indicated by the growth of the cotton plants and by comparing the roots of plants from treated and untreated areas. Roots of plants in the untreated plots were heavily galled early in the season whereas the roots of plants from the treated plots had few or no galls even at harvest time. Significant improvement in growth of the cotton plants was noted throughout the growing season in the treated blocks as compared with the untreated. However, increased growth did not result in increased yield of seed cotton. The cotton was picked late, November 10, with yields of 1474 pounds per acre from the untreated plots, 1434 pounds from the preplant plots, and 1443 pounds from the planting application plots. Marked yield increases in cotton in the San Joaquin Valley of California have been reported previously when the soil was fumigated with D-D[6] or with EDB for controlling root knot nematodes (1, 5). The reason for the lack of differences in yield in our test may be because the plants were able to utilize the nitrogen in the soil more efficiently owing to better root growth resulting from nematode control. When excess nitrogen is available, cotton sheds its squares. This results in fewer bolls, but larger plants and lower yields than are indicated by the size of the plants. These data are additionally complicated by the high incidence and severity of Verticillium wilt, also favored by soils high in nitrogen (4).

Verticillium wilt counts were made on August 27 by Stephen Wilhelm, Marvin Hoover, Extension Cotton Specialist, Alan George, and Tom Griffin of California Spray Chemical Corporation. The plants showing Verticillium wilt symptoms in 25 feet of row at each of six locations along the center two rows in each plot were counted. The total number of plants per 25 feet of row varied from 23 to 66 with a mean of 42. The percentage of Verticillium wilt was very high but the differences between treatments were not statistically significant, the percentage in the untreated being 81.29, in the preplant treatment 77.9, and in the planting treatment 78.8. Thus

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[1] Pathologist, Horticultural Crops Research Branch, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland. The senior author was Director of Research, Mid-State Chemical Supply Company, Lindsay, California when this test was made.

[2] Assistant Plant Pathologist, University of California, Berkeley, California.

[3] Farm Advisor, Tulare County, California.

[4] Mr. Harold Lembright of Dow Chemical Company supplied the ethylene dibromide, the application equipment, and assisted in this test.

[5] Dowfume W-85 was the source of the ethylene dibromide.

[6] D-D is a mixture of dichloropropene and dichloropropane.

we can conclude that in this test EDB at a nematocidal dosage did not control Verticillium wilt in cotton, nor did the control of nematodes result in a decrease in the incidence of Verticillium wilt in cotton. This is in direct contrast with the results reported from similar tests with the Fusarium wilt-root knot complex in cotton.

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WIDESPREAD AND SEVERE OUTBREAK OF TOBACCO BLUE MOLD IN THE FIELD IN 1954

L. H. Person[1] and H. R. Garriss[2]

Blue mold of tobacco, caused by Peronospora tabacina, is primarily a seed bed disease; however, it occurs sporadically in the field. The first occurrence of blue mold in the field in the United States was reported from the Gadsen-Decatur section of Florida and Georgia in 1921, the same year that the disease made its appearance in the eastern part of this country. Blue mold was absent for a period of 10 years, making its reappearance in 1931. Gaines (4) reported the field occurrence of blue mold that year in 11 counties in Georgia. In one 3-acre field, loss was estimated to be 5 percent. In most fields, however, damage was much less, as the lesions were found mostly on the lower, sand lug leaves. The number of lesions varied from a few up to 30 and even higher on some leaves. In North Carolina, Floyd (3) reported the first outbreak of blue mold in the field in 1932. The disease appeared in five counties, attacking plants about knee high.

Since these early reports, sporadic outbreaks of blue mold in the field have been reported from Florida, Georgia, South Carolina, North Carolina, Maryland, Connecticut, Massachusetts, Virginia, Wisconsin, and Canada. Every outbreak has occurred during or following periods of cool, rainy, foggy weather, conditions which are favorable for spread and development of this fungus.

In 1954, a season of light to moderate attack of blue mold in the plant bed, the most serious and widespread outbreak of blue mold developed in the field during the history of tobacco growing in the United States. The following excerpts from Warning Letters of the Plant Disease Epidemics and Identification Section describe the nature of the blue mold outbreaks in certain states: Graham, South Carolina, Letter No. 18, "There was a general outbreak of tobacco blue mold on field plants throughout the State. Some fields were observed that had 50 to 75 percent of the leaf area destroyed on the 4 or 5 lower leaves." Gilgut reported from Massachusetts, Letter No. 25, "Although there were only a few cases of blue mold in the plant beds this spring, conditions were such that spread to field plants, both Shade and Havana seed, has been more or less general." Gaines from Georgia, Letter No. 48, "Blue mold has developed to a slight extent in some Georgia fields every year since 1931. In 1954 field infections attracted more attention than usual, because the disease was rather severe only in some of the very best tobacco. Field infections were reported most frequently from May 15 to 28, and they occurred most frequently on tobacco in the lowest spots of the field and in low moist fields where growth had been rapid during April and early May. Up to 30 large necrotic spots per leaf were observed on as many as a dozen leaves per stalk, and in some instances where the crops were 5 feet high at the time, severely infected leaves occurred throughout the stalk position. In a very few isolated fields loss from these severe infections was estimated at 30 percent, but in the great majority of instances losses were negligible. Probably less than 2 percent loss was sustained by the Georgia crop."

In North Carolina, H. R. Garriss, Letter No. 15, reported "Blue mold is present in plant beds throughout the flue-cured and Burley areas. Generally, damage from blue mold in North Carolina this year has been light to date, May 14, [in the plant bed] due to lack of extended periods of favorable weather conditions for the disease. The recent cool weather has been favorable for blue mold and only lack of favorable moisture conditions has prevented severe damage in some local situations. However, even under these conditions blue mold has become reactivated in many plant beds and in a number of fields within the last few days. General rains occurred on May 14 and 15 and temperatures are very favorable for further spread and development, and considerable trouble may be expected unless clearing weather and high temperatures soon prevail."

For a week following the report of Garriss on May 14, weather conditions were very favor-

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[1] Pathologist, Horticultural Crops Research Branch, Agricultural Research Service, Raleigh, North Carolina.

[2] Pathologist, Plant Pathology Extension, North Carolina State College, Raleigh, North Carolina.

[3] Floyd, E. Y. Additional notes on the occurrence of tobacco downy mildew in North Carolina. Plant Dis. Reptr. 16: 96, 1932.

[4] Gaines, J. G. Downy mildew in Georgia tobacco fields. Plant Dis. Reptr. 15: 45, 1931.

FIGURE 1. Natural occurrence of blue mold on tobacco in the field in North Carolina in 1954.

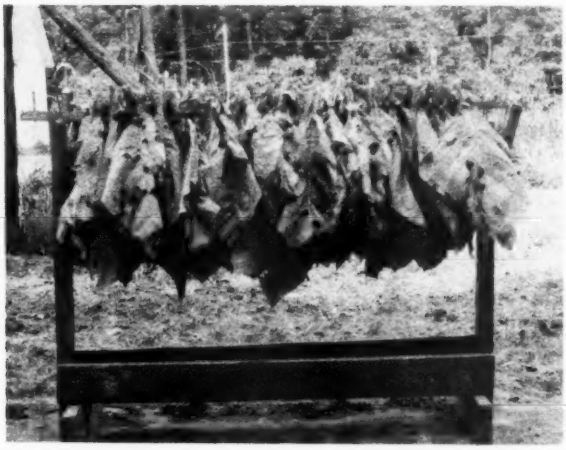

FIGURE 2. Damage on leaves of the second priming (4th, 5th and 6th leaves) caused by blue mold.

able for blue mold development and spread. From May 16 to 22 the temperature was 6 degrees Fahrenheit below long term averages and from 1 to 2 inches of rain fell over most of the tobacco area. It is evident that widespread infections occurred in tobacco fields during this period as severe leaf spot symptoms began to appear on May 18 and continued to develop until the first of June. The disease was widespread throughout most of the fields in Border and Eastern Belts, scattered in the Middle and Old Belts. During the period from May 20 to 29, 58 specimens of blue mold infection from 28 counties were received at the plant disease laboratories at North Carolina State College for diagnosis. In addition, many telephone calls were received by the Extension pathologists from County Agents and farmers concerning the severe outbreak of leaf spotting in tobacco fields.

The disease appeared most widespread in fields in which plants were from knee to waist high. In most cases leaf spotting was confined to the lower leaves, being most severe on the lower, sand lug leaves, but extending up the plant in many instances to the seventh and eighth leaf and occasionally up to the 14th leaf. Infection was general over most fields but less severe on the smaller plants (4-6 leaf stage.) On large plants (8-14 leaf stage) infection was extremely heavy on the botton 3 to 4 leaves or sand lugs (Fig. 1). Many sand lug leaves had from 30 to 50 large necrotic spots and some had up to 70 lesions which covered most of the leaf area. In many fields of large plants, the bottom 3 to 4 leaves, sand lugs or first priming, were a total loss.

This priming represents from 3 to 5 percent of the dollar value of the crop. The damage was not confined entirely to the bottom leaves as some spotting occurred on leaves higher up the stalk. Damage of the 4th, 5th and 6th leaves is shown in Figure 2. The sand lugs in this field were a total loss.

It is conservatively estimated that loss of the first priming (sand lug leaves), and in some fields damage to second and third primings, amounted to 4 1/2 to 5 million dollars.

Blue mold in the field during the season of 1954 was the most widespread and destructive during the history of tobacco growing in this State. From other reports cited above, it was also the most severe outbreak throughout the flue-cured tobacco growing area.

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TOBACCO BLUE MOLD CONTROL THROUGH PLANT BED MANAGEMENT

W. D. Valleau

During the past few years considerable progress has been made in our understanding of the life history of the blue mold fungus (Peronospora tabacina). Perhaps if we correlate the new knowledge with the old it may point the way toward plant bed practices that will lead to eradication of the fungus in the Eastern United States.

A sound program looking toward eradication is based on four points:

1. A basic difference in the method of oversummering of the fungus in its native habitat in Texas and in tobacco plant beds that are plowed before using a second time.
2. Lack of any evidence whatever that the fungus persists on wild Nicotiana species in the Southeast.
3. The fact that the fungus is known to overwinter in only two ways in the Southeast--on live plants and as oospores. Both methods can be eliminated by simple cultural practices.
4. The gradual disappearance of the fungus from an area when spore showers from farther south are eliminated.

Detailed discussion of the four points follows:

1. The fungus is endemic in Texas, where it lives on the wild species, Nicotiana repanda. In late fall when Nicotiana starts growing, the oospores, which have passed the summer in dead leaves on the soil surface, cause infection on the new rosette leaves. Conidia are formed during the winter and spring, but with warm weather sporulation ceases and oospores are formed in the dying rosette leaves. A fungus that lives through the summer in a desert climate as oospores in dead leaves on the surface of the ground might find great difficulty in surviving if the dead leaves were buried in moist soil for several months, as in a plowed bed.

2. There is no reason for believing that wild species of Nicotiana, if any are now to be found in the Gulf States, play any part in harboring the fungus. If they do, it would be expected that blue mold would develop more frequently in the Perique area of Louisiana, and that it would have appeared in Georgia and Florida before 1921 and between 1922 and 1931. The fact that it did not do so seems good evidence that the fungus probably blew in from Texas in 1921 and 1931, and that wild species are not a factor in survival and spread in the eastern United States. No one has found evidence that the pathogen is or ever was established on wild species of Nicotiana east of the Mississippi River. In the event that reinfection did occur from wild species every 10 years or so, the program to be recommended would prevent carry-over to the next year and would again eradicate it.

3. In the Eastern States, blue mold overwinters in only two ways: (a) As the sporangial or conidial stage on living tobacco plants, either suckers in the field or volunteer plants in beds abandoned the previous spring; and (b) as oospores in beds used the previous year and used again after disking or other shallow preparation. Over-wintering suckers in the field are apparently not a problem in southern Georgia. Plant beds that are not to be used a second time can readily be treated so that volunteer plants will not develop in the fall and overwinter the fungus.

Notice that normal habitat of oospores is in dead dry leaves on the surface of the ground. Person and Lucas have shown that when oospores germinate, sessile sporangia are formed on the oospores, and these produce swarm spores that cause infection on tobacco leaves in the immediate vicinity. It is necessary, therefore, that the oospores be on or very close to the surface of the ground if infection is to take place. In Kentucky we have evidence in large bed areas, extending over several years, to show that beds that are sown to soybeans following setting and plowed in the fall have no carry-over whatever. I have been informed that the same situation exists in Georgia if beds are plowed; but if they are disked, leaving the litter from the previous bed on the surface of the ground, carry-over is abundant. It is obvious that if oospore-bearing leaves are plowed under, the spores will be subjected to entirely new and different conditions from those in the native habitat and either pass out of the picture or be unable to cause infection because of being buried.

4. Evidence in Kentucky extending from 1937 to the present indicates that the blue mold fungus is not able to maintain itself year after year by oospores alone. Following blow-in of the spores, presumably from the Southeast, the fungus in succeeding years gradually disappears. Oospore infection occurs after plants are well established, the build-up is slow, and a general epiphytotic rarely develops from oospore infections alone; consequently the fungus loses ground each year. For example, following the general spore shower in Tennessee and Kentucky in 1937 the fungus completely disappeared from Kentucky by 1944 and was found in only one bed in Tennessee that year. If the fungus tends to disappear in the Tennessee-Kentucky-Ohio-Indiana area in the absence of spore showers, it should also tend to disappear in the Eastern States, provided spore showers from the area where the fungus overwinters on living plants can be eliminated. In the event that the fungus does not disappear in the Carolinas as it does in Kentucky, the use of new beds or plowing old beds should be effective in preventing carry-over.

Based on these four points, the following program should be effective in preventing carry-over on any one farm, whether in southern Georgia or any place to the north:

1. See that no suckers from the field crop go into the winter. I am assured that this is no problem in Georgia, although there has been one report of overwintering in this way; and it is not a problem farther north.
2. Use of a permanent plant bed site sown to summer cover crop soon after setting is completed, this cover crop to be plowed under (not disked) in the fall.
3. In the event the old bed site is not to be used again it should be disked, or the plants otherwise destroyed, following setting. This is to make sure the plants do not seed and produce volunteer plants on which the fungus can survive the winter and cause early infection in beds soon after plants are up.

This is a simple program and one that I understand is in line with recent recommendations throughout the flue-cured belt so far as permanent bed sites are concerned. The only added cost would be destruction of plants in beds that are not to be used again the following season.

UNIVERSITY OF KENTUCKY, LEXINGTON

NOTE: This scheme for blue mold eradication was presented January 13, 1955, at the Tobacco Disease Council meeting in Richmond, Virginia. After some discussion, nearly all favorable, the proposal was put in the form of a resolution. Seventeen members of the Council approved the resolution, and two voted against it. Signed copies of the resolution were sent to the Directors of the Florida and Georgia Agricultural Experiment Stations, for any action those States might want to take.--W. D. Valleau

CHARCOAL ROT OF CASTOR BEAN IN THE UNITED STATES

A. A. Cook

Summary

Charcoal rot, incited by Sclerotium bataticola, was found on castor bean at College Station, Texas, Blackville, South Carolina, Warsaw, Virginia, Beltsville, Maryland, and Gainesville, Florida in 1954. The causal organism, as isolated from castor bean at Gainesville, compared favorably with previous descriptions of S. bataticola ssp. typica, and was infectious on bean (Tender Green Pod), and sesame (dehiscent) as well as castor bean (U. S. 74.).

The disease commonly referred to as charcoal rot has been reported on hundreds of plants (4) from many parts of the world (2). Included among these plants is castor bean (Ricinus communis L.) which has been found infected in India (1, 3) and Palestine (2), but there has been no report of this disease on castor bean in the United States. The following is an account of the natural occurrence of charcoal rot on castor bean in southern and southeastern United States and results of preliminary laboratory and greenhouse experiments.

NATURAL OCCURRENCE AND SYMPTOMATOLOGY

During the summer of 1954, occasional castor bean plants affected by charcoal rot (Sclerotium bataticola Taub.) were observed following natural infection at College Station, Texas, Blackville, South Carolina, Warsaw, Virginia, and Beltsville, Maryland[1]. At Gainesville, Florida, at least 100 of 382 experimental plantings exhibited one or more plants affected naturally by this disease.

FIGURE 1. Section of castor bean stem just below inflorescence showing numerous sclerotia of S. bataticola.

The above-ground symptoms on castor bean are indistinct until mid-season, by which time infection has usually become well established. About this time, the stem at or about ground level may become discolored, usually blackened. Subsequent disease development under favorable environmental conditions leads to early death of the plant, still without pronounced external symptoms other than "early maturity." If, however, the roots and lower stem are examined, multitudes of tiny black sclerotia can be found beneath the epidermis, and may extend upward as far as the primary raceme (Fig. 1). Upon splitting the stem, sclerotia commonly can be found in abundance in the pith.

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[1] Personal communication from Dr. C. A. Thomas, Tobacco and Field Crops Research Branch U. S. Department of Agriculture.

GROWTH IN CULTURE

The causal organism isolated from castor bean was grown in 2% potato-dextrose-agar in petri culture plates at eight temperatures (4°C intervals) ranging from 12° to 48°C (Fig. 2). Sclerotial production was abundant between 28° and 40° after 60 hours growth with 32° the apparent optimum temperature for such production. A few sclerotia were noted at 24°.

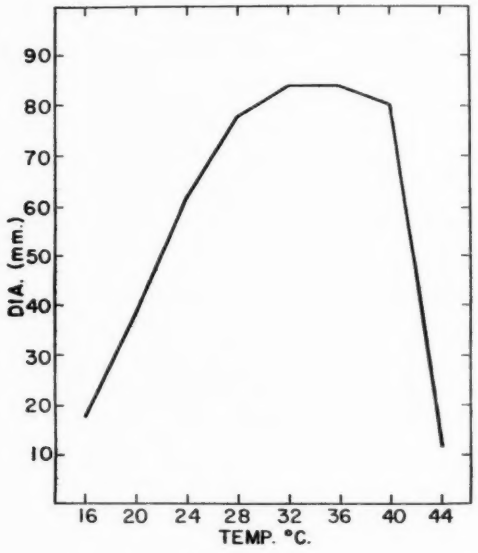

FIGURE 2. Effect of temperature on growth in culture of S. bataticola.

The isolate of the causal organism used in the studies herein reported was obtained from naturally infected castor bean plants growing at Gainesville, Florida. Its appearance in culture closely resembled the description given for S. bataticola ssp. typica by Reichert and Hellinger.

HOST RANGE

Although the natural host range of S. bataticola appears quite extensive from previous reports, artificial inoculations have not been successful with all hosts (2). In a preliminary experiment, castor bean seed (U. S. 74) was planted in steam-sterilized soil in greenhouse pots and approximately 100 ml. of a concentrated suspension of sclerotia (in water) from petri dish cultures added to each pot. One month after planting, the underground stems of half of the plants were injured by pricking with a sterile needle. Two months after planting, the roots of all 48 plants were examined under the dissecting microscope and infection diagnosed by the presence of distinct sclerotia in the roots of 96 percent of the plants, irrespective of previous treatment. Laboratory isolation from these diseased roots confirmed the visual evaluation.

In subsequent experiments, bean (Tender Green Pod), and sesame (dehiscent) were found susceptible after being planted and inoculated as described above.

DISCUSSION

A possible explanation for the occurrence of this disease in such proportions on castor bean at Gainesville during the summer of 1954 may be in part the weather. Extended hot, dry weather continued from June through August, following a normal moist, warm spring. This is not offered to add to the conflicting evidence which has been reported concerning environmental conditions prerequisite for charcoal rot. However, from the success obtained in greenhouse experiments, where no emphasis was placed on maintenance of any particular environment, and from natural occurrence in the field, it would appear that the influence of environment on charcoal rot infection and development may well be investigated further.

The illustration by Thirumalachar of S. bataticola ssp. sesamica, reported by that author as having been isolated from castor bean, does not resemble the isolate from that host which was used in these experiments. Furthermore, the isolate used in these studies closely resembles the description of S. bataticola ssp. typica by Reichert and Hellinger as well as that illustrated by Thirumalachar as sub-species typica.

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FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, IN COOPERATION WITH THE AGRONOMY DEPARTMENT, AGRICULTURAL EXPERIMENT STATION, GAINESVILLE, FLORIDA.

A LEAFSPOT OF LOTUS ULIGINOSUS CAUSED BY CERCOSPORA LOTI[1]

K. W. Kreitlow and Helen Sherwin Yu[2]

Summary

Cercospora loti, a fungus reported previously only from Hungary, was found causing a leafspot of Lotus uliginosus in Maryland and in Georgia. Pathogenicity of the fungus was established on L. uliginosus, which appeared to be very susceptible in comparison with L. corniculatus and L. tenuis. The fungus was capable of attacking the stems of inoculated plants ultimately causing death of diseased shoots.

A leafspot of the forage legume Lotus uliginosus Schk. caused by a Cercospora hitherto not reported on this host was found at Beltsville, Maryland, and near Alapaha, Georgia, in 1954. According to Chupp (1) Cercospora loti Hollós was described by Hollós on leaves of L. siliquosus L. in Hungary. A search of the literature revealed no additional reports of this or any other species of Cercospora attacking Lotus spp.

The leafspot was first noticed June 4, 1954, causing some defoliation among two-year-old plants in a clonal nursery at Beltsville, Maryland. On June 15, a similar leafspot was observed on leaves of L. uliginosus in a piney woods pasture near Alapaha, Georgia. At both locations, many lower leaves were infected and fell off at the slightest touch.

Typical symptoms of infection are shown in Figure 1. Usually only one large spot develops on a leaflet. Each leafspot is characteristically orbicular, avellaneous to dark olive (3) and up to 1 cm. in diameter, averaging 3-5 mm.

Fruiting is both epiphyllous and hypophyllous. The dense fascicles of conidiophores are borne on a stroma. The conidiophores are pale olivaceous brown to olive gray and are 1-5 septate. They are geniculate with 1-4 spore scars, unbranched, and measure 3.6-5.9 x 21-108 µ. Conidia are hyaline, straight to slightly curved, usually truncate at the base and tapering slightly to a sub-acute tip. The conidia are indistinctly multiseptate (up to 27 septa being counted in a spore) and measure 3.3-5.9 x 22-224 µ.

The shape and size of conidia varied considerably among collections made at intervals throughout the growing season at Beltsville, Maryland. On diseased leaves collected in June, conidia were relatively short and wide with few septa. The conidiophores also were short with few septa or spore scars and they were noticeably bulged. On diseased leaves collected in October, the conidia were longer, narrower, and had more septa. The conidiophores also were longer, had more septa, more spore scars, and were not bulged. New infections occurred at Beltsville until November 1.

Single-spore isolates of the Cercospora cultured on potato-dextrose agar produced a dark grayish-olive growth (3). Sporulation was sparse on agar. In carrot decoction plus dextrose, growth was slow but sporulation was more abundant.

Inoculum for testing pathogenicity of the Cercospora was prepared by growing several single-spore and multi-spore isolates from the Maryland and Georgia collections in 250 ml. Erlenmyer flasks containing 35 ml. of carrot decoction plus dextrose. After 14 days' growth, the cultures were macerated in a Waring blendor and the isolates from Maryland and Georgia were sprayed separately on duplicate sets of ten plants each of Lotus uliginiosus, L. corniculatus and L. tenuis, which had been growing in the greenhouse for 7 weeks. Two sets of check plants of each species were sprayed with sterile distilled water.

All plants were incubated in a moist chamber at 25°C for 72 hours. One set of inoculated plants and controls was then transferred to a temperature-controlled room held at 18.5° under a 12-hour daylength. The other set was incubated in a greenhouse at 29.5°. The plants at each temperature received supplementary moisture at night from fog-producing humidifiers to simulate heavy dew.

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[1] The authors are indebted to Dr. Charles Chupp, Department of Plant Pathology, Cornell University, Ithaca, New York, who examined specimens from Maryland and Georgia and confirmed identification of this Cercospora.

[2] Senior Pathologist and Assistant Pathologist, respectively, Field Crops Research Branch, Agricultural Research Service, United States Department of Agriculture.

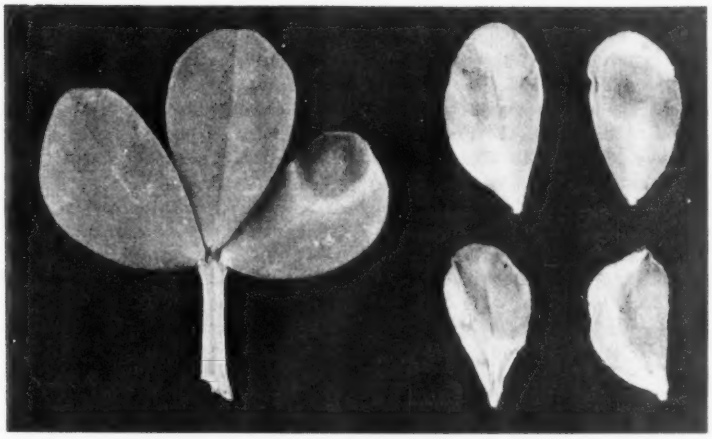

FIGURE 1. A trifoliate leaf and leaflets of Lotus uliginosus. Leafspot of Lotus uliginosus caused by Cercospora loti.

Lesions similar to those seen in the field developed on leaves of inoculated plants of Lotus uliginosus within 6 days at 29.5°C. Comparable infection developed at a slower rate on leaves of inoculated plants incubated at 18.5°. When the lesions were about 1mm. in diameter, they were light tannish-gray with a slight purple tinge. They enlarged rapidly and at the end of 12 days were olive-gray to reddish-brown. Conidia of Cercospora developed profusely in the lesions. Within a short time diseased leaves usually shriveled and dropped. Reddish-brown lesions frequently extending several centimeters also developed on stems and ultimately caused death of diseased shoots. No infection developed on check plants.

Only a few, small, reddish-brown lesions ultimately developed on leaves of inoculated plants of L. corniculatus. Nearly three weeks were required for spots to develop and only a few of the lower leaves dropped off. Lotus tenuis appeared to be highly resistant to infection since only a few lesions developed on leaves of inoculated plants. When leaves of L. corniculatus and L. tenuis bearing suspected lesions were placed in Petri dish moist chambers, conidia of Cercospora were produced in the spots. No lesions developed on leaves of the check plants.

There was some indication that the isolates from the Georgia material were more pathogenic than the Maryland isolates. Both, however, were highly pathogenic to Lotus uliginosus.

Characteristics of the Cercospora attacking L. uliginosus in the United States resemble those described by Hollós (2) for Cercospora loti attacking L. siliquosus in Hungary. Whereas Hollós reported that fruiting is only epiphyllous we find it both epiphyllous and hypophyllous. The conidiophores are 1- to 5-septate instead of 1- to 2-septate as he stated and conidia and conidiophores are longer and wider than he described. Since environmental conditions are known to influence size of conidia and conidiophores the dissimilarity in measurements is probably not significant. None of Hollós' material was available for comparison; however, it is felt that from microscropic characteristics of conidia and conidiophores and from pathogenicity tests on three species of Lotus the fungus causing the leafspot of L. uliginosus is Cercospora loti. It seems strange that the fungus has not been reported since Hollós' original descriptions nearly 50 years ago. The occurrence of the disease in two widely separated parts of the United States suggests that it may occur elsewhere on this host and has probably been overlooked because of the destructive effect of foliar blight caused by Rhizoctonia solani. There is a possibility that the disease may have been introduced on imported seed; however, nothing is yet known concerning seed transmission of Cercospora loti. Additional studies on the host range and distribution of the pathogen are underway.

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DISEASES OF FORAGE LEGUMES IN IOWA IN 1954

John W. Baxter

ALFALFA: The most important diseases of alfalfa in Iowa during 1954 were: spring black stem (Ascochyta imperfecta), summer black stem and leaf spot (Cercospora medicaginis), common leaf spot (Pseudopeziza medicaginis), Stemphylium leaf spot (Stemphylium botryosum), bacterial wilt (Corynebacterium insidiosum), and Leptosphaeria leaf spot (Leptosphaeria pratensis). The last-named disease, which has not previously been reported from the State on alfalfa, was prevalent in western and central Iowa during July and early August.

Diseases of minor importance on alfalfa were: root rot (Fusarium spp.), rust (Uromyces striatus var. medicaginis), downy mildew (Peronospora trifoliorum), yellow leaf blotch (Pseudopeziza jonesii), and anthracnose (Colletotrichum destructivum).

The blackstem complex was present throughout the season and appeared to be the most destructive of the diseases on alfalfa. Isolations from blackened stems during late summer frequently yielded the anthracnose organism, Colletotrichum destructivum, although typical anthracnose symptoms were absent. Isolates of Cercospora medicaginis were obtained from blackened stems and leaf lesions throughout the summer and as late as the first week in October. In cross-inoculation experiments Cercospora isolates from alfalfa, red clover, and sweetclover infected only the host from which they had been obtained.

RED CLOVER: The root rot complex (Fusarium spp. and other fungi) was the most important disease of red clover in 1954. Second only to root rot in importance was angular leaf spot and blackstem (Cercospora zebrina), which appeared in early June and was present in moderate to heavy amounts until late September. Northern anthracnose (Kabatiella caulivora) was abundant during late June and early July. Diseases of minor importance on red clover were: rust (Uromyces trifolii), powdery mildew (Erysiphe polygoni), Stemphylium leaf spot (Stemphylium botryosum), spring black stem (Phoma trifolii), sooty blotch (Cymadothea trifolii) and southern anthracnose (Colletotrichum destructivum).

SWEETCLOVER: Diseases of importance on sweetclover were: spring black stem (Ascochyta lethalis), summer black stem (Cercospora davisii), stem canker (Ascochyta caulicola), Leptosphaeria leaf spot (Leptosphaeria pratensis) and anthracnose (Colletotrichum destructivum). Anthracnose was particularly severe on certain lines of sweetclover in experimental plantings at Ames during August and September.

BIRDSFOOT TREFOIL: Rhizoctonia foliage blight (Rhizoctonia solani) was a serious disease in experimental plantings of birdsfoot trefoil at Ames and Albia. The pathogen was isolated from blighted leaves, stem cankers and rotted crowns.

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FOOT ROT OF POTHOS, A DISEASE CAUSED BY RHIZOCTONIA

D. F. Millikan and J. E. Smith, Jr.[1]

Abstract

In 1954 a severe epiphytotic was observed on Pothos aureus. The first symptoms are a loss in turgidity of the leaves followed by a dark brown to black lesion at the soil line. Presumably, this is preceded by a rotting of the major roots. A Rhizoctonia sp. consistently was found associated with the disease, and experimentally induced symptoms similar to those observed under greenhouse conditions. The use of sterilized soil apparently will control the disease.

For many years the greenhouse industry of Missouri has used non-sterilized soil in their normal cultural practices. Although such practices encouraged losses due to the damping-off organisms, the losses were usually light and insufficient to warrant the installation of equipment necessary to eliminate them. In the spring of 1954, however, a severe epiphytotic similar to that described on Philodendron[2] broke out in a commercial establishment specializing in the propagation and sale of Pothos aureus, a popular variegated houseplant. Losses upwards of 30 percent were commonly experienced, serious enough to make the profitable growing of this crop questionable.

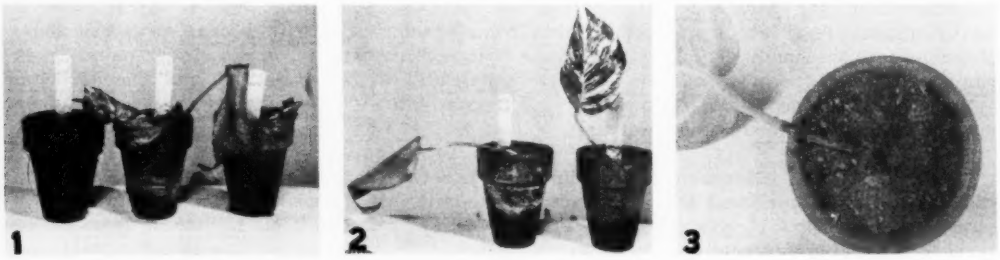

FIGURE 1. Rhizoctonia foot rot of Pothos aureus. Left, plant showing initial symptoms of loss in turgidity. Center, plant completely girdled at soil line. Right, plant completely dead.

FIGURE 2. Left, diseased plant in non-sterile, uninoculated soil. Right, healthy plant in sterile soil.

FIGURE 3. Closeup of diseased plant, showing lesion at soil line.

The Disease

This disorder appears 1 week to 10 days after the cuttings are transplanted from the rooting beds into pots. The first suggestion of the disease is a loss of turgidity of the leaves, producing a noticeable limp or flaccid condition. This is followed by a blackening of the main stem at the soil line that may form a lesion about 1/2 inch long, usually sufficient to girdle the stem. Vessels of the affected stem appear to be filled with an increased amount of gumming. Examination of the affected plants suggests that the earliest symptoms are the blackening and necrosis of the roots extending as far back as the main stem. Initial isolations revealed a bacterium and a fungus, neither of which proved to be pathogenic. However, when the isolation technique was

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[1] Plant Pathologist and Associate Professor of Floriculture, respectively.

[2] French, Alex M., and Carl Nichols. 1954. Sclerotium rolfsii on Philodendron cordatum in Southern California. Plant Dis. Reptr. 38: 530.

modified according to Luttrell[3] Rhizoctonia was consistently obtained, and occasionally a species of Fusarium. This technique consists of a very mild washing with tap water to remove the soil fragments and plating directly to 2% water agar. In studies with both organisms artificial inoculation with Rhizoctonia readily duplicated the disease as it was observed in the greenhouse. Furthermore, an examination of the diseased tissue revealed the presence of the organism in the rotted plant tissues.

Inoculation Studies

The first experiment was set up on December 8 with 25 plants divided into five different treatments. Five plants each were potted into sterile soil (S), non-sterile soil (NS), sterile soil plus Rhizoctonia (S + R), sterile soil plus Rhizoctonia plus Fusarium (S + R + F), and sterile soil plus Fusarium (S + F). Both organisms were grown on sterile oats and used after about 2 weeks by mixing the inoculum with the soil. The results of this experiment are listed in Table 1.

Table 1. Incidence of foot rot in Pothos aureus plants grown in uninoculated sterile (S) and non-sterile (NS) soil and in sterile soil inoculated with Rhizoctonia (R) and/or Fusarium (F).

Number of plants	Soil treatment	Foot rot incidence[a] Number of plants		
		Unaffected	Wilted	Dead
5	S	5	0	0
5	NS	4	0	1
5	S + R	0	0	5
5	S + R + F	2	0	3
5	S + F	5	0	0

[a] Readings taken 10 days after potting.

The results indicated that Rhizoctonia was associated with the disease noted in the greenhouse. Inasmuch as Fusarium alone failed to cause the disease while pure cultures of Rhizoctonia did, it seemed safe to eliminate the former organism as a major factor in the diseased condition. Furthermore, the isolation of Rhizoctonia from the diseased plant in the non-sterile uninoculated soil supports this premise.

A second experiment was set up on December 22. The results listed in Table 2 tend to confirm those of the earlier experiment, although not quite so striking. The data show the role of Rhizoctonia in the disease complex. In addition they indicate that the disease can be controlled by using sterilized soil.

Table 2. Incidence of foot rot on Pothos aureus grown in sterile (S) and non-sterile (NS) soil, with and without Rhizoctonia inoculum.

Number of plants	Soil treatment	Foot rot incidence[a] Number of plants		
		Unaffected	Wilted	Dead
8	S	8	0	0
8	NS	8	4	0
11	S + R	5	2	4
10	NS + R	5	3	2

[a] Readings taken 10 days after transplanting.

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[3] Luttrell, E. S. Private communication.

FASCIATION OF ROOTS IN GALLBERRY

J. H. Owen

Fasciated roots of gallberry, Ilex glabra L. were recently sent to Gainesville, Florida for examination. The flattened and fan-shaped roots were found by R. L. Reese of the Ft. Pierce, Florida Field Laboratory (Fig. 1). The affected plant was growing on open land that had been

FIGURE 1. Fasciated roots of gallberry, Ilex glabra L. Smaller rootlets appeared to be normal.

cleared in 1952 and prepared for citrus planting. A regrowth of gallberry had occurred and was being cleaned out when the fasciated roots were observed. This was the only plant having this type of roots, and approximately 65 to 75 percent of the roots were fasciated. The above-ground portions of the affected plant appeared to be normal, and apparently the fasciated roots produced no visible variation. The plant was growing on soil classified as Leon sand with hard-pan at depths of 24 to 36 inches. No weed killing chemicals had been used on this land.

Fasciation of aerial parts of plants is of common occurrence, and fasciated aerial roots of cacti and orchids have been reported[1]. A search of the literature failed to reveal any information concerning fasciation of subterranean roots.

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[1] White, O. E. 1948. Fasciation. Bot. Rev. 14: 319-358.

SPOT ANTHRACNOSE OF CHINESE HOLLY[1]

A. G. Plakidas

In January, 1947, several Chinese holly trees (Ilex cornuta Lindl.) were found at Hammond, Louisiana, with numerous black spots on the upper surfaces of their leaves. Spotted leaves were collected and a Sphaceloma (Elsinöe) was readily isolated from the black lesions. Subsequent examinations of these trees revealed other symptoms, namely, bark lesions on the shoots (Fig. 1, C), scabby lesions on the berries, and a large, elongated, dry, grayish black, scabby lesion occurring usually on one-half of the leaf blade and causing a severe distortion of the leaf (Fig. 1, B). The Sphaceloma was isolated many times from the lesions on the leaves, shoots, and berries. A technical description of the disease and its causal organism has been published[2]. The present paper is written primarily for the convenience of the holly growers and others who may not have access to the technical publication.

Origin of the Disease

As far as can be determined, this is a new disease of this holly. No report of a spot anthracnose or any similar disease of this host has been found in the literature. The trees on which the disease was first observed were grown from seed that came from a tree on the Louisiana State University campus in Baton Rouge. Although the disease was later found in this tree, it seems unlikely that the disease was transmitted by means of seed. Seed transmission of spot anthracnose has yet never been reported.

As there was a possibility that the disease had spread to the trees in Hammond from some infected host, a search was made in surrounding gardens and also in the neighboring woods where Ilex opaca and I. vomitoria abound, but the search was unsuccessful. Material (leaves and shoots) with suspicious lesions was collected from both cultivated and wild hollies and cultured, but in no case was a Sphaceloma isolated.

Description of the Disease

Two distinct types of symptoms occur on the leaves. For convenience these types are designated as type A and type B.

Type A, the more prevalent of the two, is characterized by very numerous black spots. These spots, which at first are very small, gradually enlarge to 1-2 mm in diameter. They often coalesce and form irregular black patches (Fig. 1, A) which sometimes cover as much as 50 percent of the leaf surface. The spots occur, for the most part, on the apical one-half of the leaf, but occasionally may be evenly scattered over the entire leaf surface. They are confined to the upper surface of the leaf, and the injury to the tissue is limited to the upper epidermis and the palisade cells. The lower side of the leaf, directly below the lesions, appears normal.

Type B, (Fig. 1, B) is characterized by a large lesion, often about 1 1/2 inches in length, which typically is confined to one-half of the leaf blade. More than one lesion has occasionally been observed on the same leaf. Since growth of the tissues on the affected side is not normal, the leaf becomes markedly distorted in shape. As in type A, the lesions of type B also are confined to the upper surface, but the injury to the tissues extends deeper and sometimes becomes visible on the under surface. In the early stages, the affected areas are more or less yellow and marked with irregular brown patches. At this stage, the epidermis is intact. Later, the epidermis ruptures and sloughs off and the color of the lesion becomes dark gray. Still later, when most of the epidermis has disappeared, the surface of the lesion becomes rough, hard and coal black.

Type B infection is less prevalent than type A. It has been found on only three trees. One of these, a large, staminate tree, was severely affected with about one-fourth of the foliage spotted; the other two trees had only a few infected leaves. The severity of the disease on these trees did not change materially during a 5-year period.

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[1] In a preliminary report (Phytopath. 40: 22, 1954) the disease was referred to as "scab", but it seems more appropriate to accept the suggestion of Jenkins (Plant Dis. Reptr. 31: 71, 1947) and use the term term "Spot anthracnose."

[2] Plakidas, A. G. 1954. Spot anthracnose of Chinese holly. Mycologia 46: 346-353.

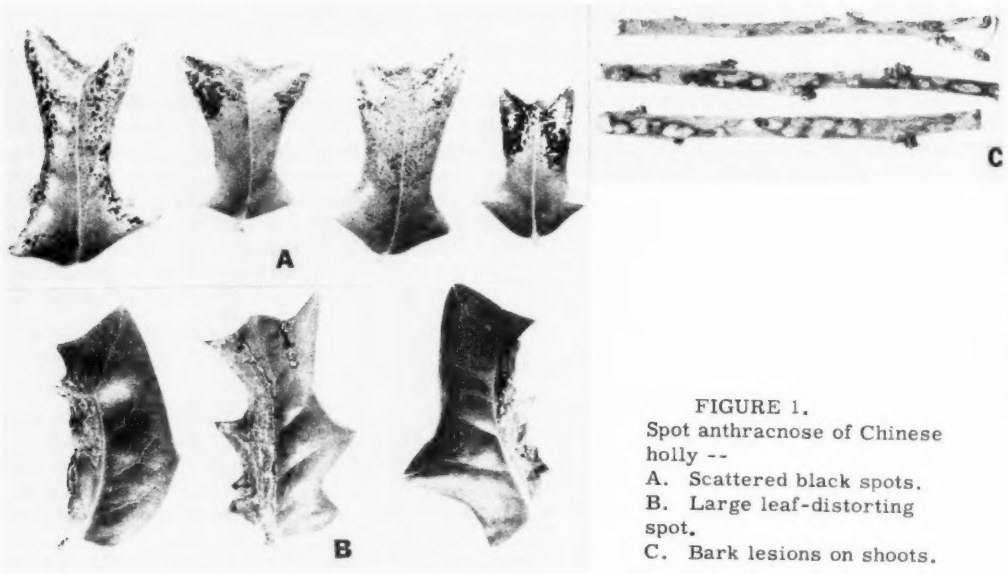

FIGURE 1.
Spot anthracnose of Chinese holly --
A. Scattered black spots.
B. Large leaf-distorting spot.
C. Bark lesions on shoots.

Another important difference has been observed between types A and B, and that is the time of the year in which symptoms appear on the new growth. Symptoms of type B appear in early summer and those of type A in late fall. During one year the new growth of affected trees was examined periodically from March until December. Type B lesions were found on the new leaves on July 11. As they were not present on May 3, when the previous examination was made, they must have developed sometime between May 3, and July 11, probably during June. In contrast, black spot lesions (type A) were abundant and well developed on December 18, but not on October 17. They apparently developed sometime between these two dates, probably during November.

It is believed that the differences between the two types can be interpreted to be a host reaction. The trees were raised from seed, and they varied considerably as to size, habit of growth, and size and shape of the leaves. It is reasonable to think that they might also react differently to the pathogen.

Actually these trees fell within three distinct groups: those completely free of disease, those showing type A foliage symptoms, and those showing type B foliage symptoms.

The bark lesions on the shoots vary considerably with age. Young lesions (Fig. 1, C) on current season's growth are small, 1/2 to about 4 mm in diameter, flat or slightly raised, light brown in color at first, but later with grayish centers and light brown margins. Often a fine longitudinal crack occurs in the center of the lesion. Older lesions (on previous season's growth) are larger and they usually coalesce to cover large areas (1/2 to about 3 inches in length) of the shoot (Fig. 1, C). The margins of the older lesions are dark brown, and the centers gray. Numerous minute black specks (acervuli, the spore-producing organs of the fungus) occur on the gray portions of the lesions. Finally, the appearance of the older lesions is complicated by the growth of secondary fungi which invade the injured and dead tissue and also by that of the alga, Cephaleuros virescens, which often grows profusely on the affected bark.

The lesions on the green berries begin to appear in late June and become very prominent by the middle of July. They start as small brown spots, flat or slightly raised, later becoming corky, raised, and cracked. The cracks are sometimes fine, hardly visible to the naked eye, but may be large and prominent.

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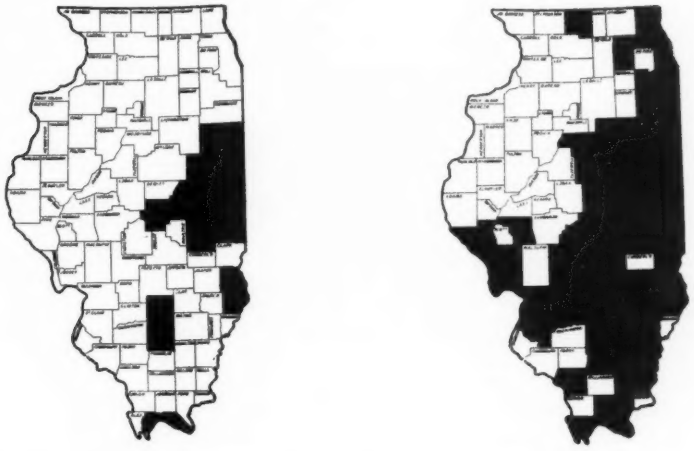
SPREAD OF DUTCH ELM DISEASE IN ILLINOIS IN 1954

R. J. Campana and J. C. Carter

The Dutch elm disease (Ceratostomella ulmi) has spread extensively into northern and western Illinois and has increased in southern and eastern parts of the State during 1954 (Fig. 1). From the summer of 1950[1] to that of 1954[2] at least 532 cases of the disease had occurred in 32 localities in 15 counties (Table 1). In 1954 the number of diseased trees increased to such a degree that an accurate count of them throughout the State is no longer possible. During this year 2,067 trees (Table 2) were tallied as diseased; this figure is based on culture records, field diagnoses, and counts made in the worst affected areas. A conservative estimate of the actual number of diseased trees in the entire State would be considerably higher. In a single roadside survey of 50 miles through intermittent and sparsely-wooded areas in east central Illinois, 72 diseased trees were tallied. This survey indicates the probability that many trees are affected with Dutch elm disease which are unknown and undetected in other wooded areas. Figures on numbers of diseased trees represent only counts within communities in most cases.

Presence of the disease has now been confirmed by laboratory culture for 77 new localities in 40 additional counties affected in 1954, bringing the total numbers for a 5-year period to 111 localities in 55 counties (Table 2). Counties newly affected with Dutch elm disease in 1954 and the numbers of cases for each county are listed in Table 3.



1953 1954

FIGURE 1. Distribution by counties of Dutch elm disease in Illinois in 1953 and 1954, indicating spread during 1954.

From the known distribution of the disease, in all but 17 counties (Fig. 1), south and east of the Illinois River, an area comprising about two-thirds of the State, it is suspected that the disease may occur in all of this area. Of special interest and great concern to the people of the areas affected is the occurrence of the disease as far north as Rockford in Winnebago County, about 15 miles from Wisconsin and about 70 miles from Iowa, as well as in four of five counties in the Chicago area. In the Chicago area diseased trees were located in counties as follows: 1 at Highland Park in Lake; 1 at Aurora in Kane; 2 each at Joliet and Peotone in Will; and 1 at Markham in Cook.

The disease was found as far west as Kampsville in Calhoun County and Milton in Pike

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[1] Carter, J. C. 1950. Dutch elm disease in Illinois. Plant Dis. Reptr. 35: 56.

[2] Campana, R. J. 1954. The present status of Dutch elm disease in Illinois. Plant Dis. Reptr. 38: 356-358.

Table 1. Dutch elm disease recorded for counties in Illinois.

Counties	Numbers of diseased trees					
	1950	1951	1952	1953	1954	Totals
Champaign	0	4	17	183	825	1029
Coles	1	1	0	233	600	835
Crawford	0	0	0	1	0	1
Douglas	0	5	2	42	75	124
Edgar	0	0	0	9	0	9
Ford	0	0	0	1	71	72
Iroquois	0	0	0	2	40	42
Jefferson	0	0	0	1	0	1
Lawrence	0	1	0	2	0	3
Macon	0	0	2	0	0	2
Marion	0	0	1	15	0	16
Massac	0	0	1	5	2	8
Piatt	0	0	0	1	20	21
Pulaski	0	0	1	0	0	1
Vermilion	0	0	1	0	9	10
Totals	1	11	25	495	1642	2174
Cumulative totals	1	12	37	532	2174	

Table 2. Five year Dutch elm disease record for Illinois.

Year	Numbers of new cases[a]	Numbers of localities affected			Numbers of counties affected		
		Old	New	Total	Old	New	Total
1950	1	0	1	1	0	1	1
1951	11	0	5	5	1	3	4
1952	25	2	8	10	2	5	7
1953	495	8	20	28	6	6	12
1954	2067	14	77	91	8	40	48
Totals	2599		111			55	

[a] At least one confirmation by culture for each new county and for all but very few localities.

County, about 10 and 20 miles, respectively, from the Missouri boundary. With the rapid spread of the disease north and west over Illinois during 1954, and with its proximity to relatively nearby areas of Wisconsin and Iowa, its invasion of these States in 1955 is a serious possibility. It was reported in Missouri as early as 1952.

As expected, greatest increases in numbers of diseased trees in 1952 occurred in counties where the disease had been most abundant in previous years (Table 1). Counties worst affected had the following number of new cases recorded: Douglas 75; Champaign 825; Coles 600; Ford 71; Iroquois 40. In seven (Crawford, Edgar, Jefferson, Lawrence, Macon, Marion, Pulaski) of 15 counties with diseased trees in previous years no new cases were reported. Since it was not possible to survey these areas systematically for the disease, new cases there may have occurred undetected, or have been attributed to the drought, which was severe in all such areas.

In most counties newly affected by the disease in 1954 (Table 3), there were relatively few cases recorded. But in several counties the numbers of diseased trees and their distribution were sufficient to indicate abundant sources of Dutch elm disease for serious increases in future years. Counties worst affected had the following numbers of cases: Clay 20; DeWitt 22; Kan-

Table 3. Counties[a] newly affected with Dutch elm disease in 1954.

County	Number of cases	County	Number of cases
Alexander	1	Kane	1
Bond	1	Kankakee	240
Calhoun	1	Lake	1
Christian	1	Livingston	62
Clay	20	Madison	1
Clark	1	McLean	26
Clinton	6	Montgomery	1
Cook	1	Morgan	1
DeWitt	22	Moultrie	3
Edwards	1	Pike	1
Effingham	1	Pope	1
Fayette	2	Richland	3
Franklin	1	Saline	2
Gallatin	1	Shelby	1
Greene	1	St. Clair	1
Hamilton	1	Wayne	2
Jackson	1	White	2
Jasper	1	Will	8
Jersey	1	Winnebago	1
Johnson	1	Woodford	1
Total	66		359
Grand Total			425

[a] First year of detection and confirmation by culture.

Table 4. Incidence of elm phloem necrosis and Dutch elm disease in Champaign-Urbana.

Year	Numbers of diseased trees		Percent[a] of Dutch elm disease
	Phloem necrosis	Dutch elm	
1944	2	0	0
1945	1	0	0
1946	2	0	0
1947	3	0	0
1948	8	0	0
1949	99	0	0
1950	313	0	0
1951	359	1	0.55
1952	555	11	1.96
1953	388	164	29.71
1954	179	694	79.49
Total	1909	870	

[a] Percent of diseased trees affected with Dutch elm disease.

kakee 250; Livingston 62; and McLean 26. The unusually large number of cases in Kankakee County found mostly in and around the city of Kankakee is attributed to presence of the disease there at least a year previous to discovery. Although not reported or cultured before 1954, the Dutch elm disease fungus was detected in the 1953 wood of some trees after the disease had become conspicuously epidemic in 1954.

In the Champaign-Urbana area the incidence of Dutch elm disease has been studied closely with special reference to the incidence of the virus disease elm phloem necrosis in the same area. In this area Dutch elm disease was preceded by phloem necrosis by 7 years (Table 4). In 1951, the first year Dutch elm disease occurred there, it killed less than 1 percent of the trees affected by both diseases. Its percentage of mortality has increased successively, however, from about 2 percent in 1952 to 30 percent in 1953 and 79 percent in 1954. The rapid build-up here of Dutch elm disease until it out-stripped phloem necrosis is similar to that reported by Whitten[3] for Bexley, Ohio. In Champaign-Urbana the unusually rapid increase of Dutch elm disease is attributed to the tremendous build-up in population of the European elm bark beetle. This has resulted from the presence of large numbers of dead or dying elm trees which were not removed. There is evidence that some trees apparently dying of Dutch elm disease in Champaign-Urbana are also infected with phloem necrosis. Of ten trees known to be affected with phloem necrosis in 1953, at least one had Dutch elm disease in 1954.

The spread and intensification of Dutch elm disease in the southern half of Illinois is attributed primarily to a tremendous increase in population of the European elm bark beetle in elm wood from trees dead and dying of phloem necrosis and Dutch elm disease. Northern extension of the disease beyond the phloem necrosis zone was probably facilitated by climatic conditions which favored increase of the bark beetle. Thousands of elm trees throughout Illinois were observed to be suffering from drought in the summer of 1953 and 1954. Many of these trees when examined were heavily infested with bark beetles, and some mortality occurred. No evidence of disease could be found in many trees so affected. Illinois had one of the mildest winters on record in 1953 and 1954. This probably made possible the survival of countless millions of bark beetles which never would have survived a normal winter.

SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, ILLINOIS STATE NATURAL HISTORY SURVEY DIVISION, URBANA

[3] Conference Report on Dutch elm disease, elm phloem necrosis, oak wilt, N. Y. City, Feb. 4-5, 1954.

NATURAL SPREAD OF ENDOCONIDIOPHORA FAGACEARUM BRETZ TO WOUNDED RED OAKS IN IOWA[1]

Dale M. Norris, Jr.

Summary

The results of several studies on the role of wounds in natural overland transmission of Endoconidiophora fagacearum Bretz in Iowa are reported and discussed. Natural spread occurred in 31 of 122 red oak trees which were wounded during the period from April 24 to June 22. Only 2 of 217 red oak checks wilted; these were wounded by rodents during the test period and wilt symptoms developed progressively from the injured area on each tree. The highest percentage of infection occurred in trees wounded during the interval from May 11 to 22 inclusive. A single wounded red oak located in an otherwise oak wilt-free group of red oaks attracted vectors, apparently insects, carrying sufficient inoculum to infect and kill it. Such overland spread occurred in five of six experimental areas.

Introduction

The importance of wounds in the overland spread of Endoconidiophora fagacearum Bretz from diseased to isolated healthy oaks has been strongly suspected in Iowa since the current "long distance" dissemination studies were initiated during the summer of 1952. The first isolated wilt-infected oak observed by the author bore a large sappy wound at the base of the only limb showing symptoms (July 31, 1952). One adult specimen of the nitidulid beetle, Glischrochilus quadrisignatus (Say), was found feeding on this wound. The oak wilt fungus was never successfully isolated from this beetle, but the fact that this species and many other sap- and fungus-feeding insects have been found so closely associated with the sporulating oak wilt fungus and later with many other wounds on healthy and isolated wilting oaks made them the foremost of suspected vectors. Nearly 100 percent of the isolated wilting oaks that have been seen since have borne a conspicuous wound somewhere on the trunk or major limbs; frequently the active symptoms have been limited to that portion of the tree which is distal to the wound.

The first published information on the possible role of wounds in the overland spread of the oak wilt fungus was by Guyton (6) in Pennsylvania. In this instance, oak wilt killed a number of oaks which had been blazed to mark a future logging road. The possible role of wounds in "long distance" transmission was later suggested by Craighead and Morris (1).

The apparent importance of wounds in "long distance" spread was first demonstrated experimentally by Dorsey et al. (3) and Norris (7) in simultaneous reports of successful insect transmission of Endoconidiophora fagacearum through wounds. An account of transmission of the oak wilt fungus to a wounded oak seedling by the pomace fly was later published by Griswold (4). Further substantiating data on the apparent necessity of wounds for overland dissemination were added by Craighead, Morris, and Nelson (2). In this work, 194 trees were wounded periodically from March 23 to July 31, 1953; of these 8 trees wilted. This work showed that other factors being favorable, overland spread did occur when the proper wound conditions existed in healthy oak trees.

Beginning in April, 1954 and continuing at the present time, tree wounding experiments have been conducted in the State parks located at Forest City and Lehigh, Iowa. The general purpose of these investigations has been to determine more clearly the exact nature and extent to which wounds are involved in overland spread under natural conditions. In these studies all aspects of overland transmission of oak wilt, except providing the wounds in the healthy trees, have been left to nature.

[1] Journal Paper No. J-2698 of the Iowa Agricultural Experiment Station, Ames, Iowa. Project No. 1047. Research sponsored jointly by the National Oak Wilt Research Committee, the Iowa State Conservation Commission, and the United States Department of Agriculture.

Methods

These investigations were conducted on red oak trees in Pilot Knob State Park, Forest City, Iowa; or Dolliver Memorial State Park, Lehigh, Iowa. The trees ranged from approximately 6 to 18 inches d.b.h.; however, within any one experiment an effort was made to select trees of similar d.b.h. All trees were beyond possible root graft-infection distances from diseased trees. Several experiments involving from five to six trees up to several hundred trees were conducted. The two major studies involved periodical woundings of plots of trees at intervals of 10 to 15 days. With one exception, the smaller investigations involved only one wounding date.

The wounds were T-shaped or chopped areas in the bark as described by Norris (7). In both types, the bark was left covering as much of the wound as possible. Three or four wounds were made on each tree. These were located at heights ranging from 3 to 4 feet up to 15 to 20 feet, and were made on the main trunk or a major limb of the tree. All wounds were made with either a hatchet or large knife; the blade of which had first been surface sterilized in a 100% Clorox solution.

Positive oak wilt infection in trees involved in wounding studies was confirmed by laboratory culture from the infected wood.

In discussing the experiments reported in this paper, each one will be given a number and referred to subsequently by this number in an effort to maintain greater clarity. On April 20, 1954 at Dolliver Memorial State Park, experiment I, the first of two major tree-wounding studies, was initiated. From this date on ten red oak trees were wounded at each 10- to 15-day interval throughout the spring, summer, and fall. These trees were located in a 30-acre area of oak-hickory woods which was free of oak wilt, but surrounded on all sides by pockets of diseased and dead oak trees. The trees of each plot were located at random throughout the entire area in an effort to minimize any effect which their relative positions with regard to inoculum sources might have on the amount of infection.

The second major study, experiment II, was begun at Forest City, Iowa on April 24, 1954. The experimental area in this case was approximately 10 acres in size and was located within one of the few oak wilt-free areas of this park. All test trees were within 1/2 mile of 200-300 wilt-killed oaks which provided abundant inoculum when mats and pads were being produced. Eight trees were wounded at each 10- to 15-day interval in this study.

Several smaller tree-wounding studies were also conducted. In experiment III, five red oaks were wounded around a lone inoculum-bearing oak at Pilot Knob State Park on May 7, 1954. The wounded trees were located in a ring 150 feet from the diseased tree. Five other oaks situated at a similar distance from the dead tree were marked as checks. The wounds on each of the five trees in this experiment were extended into fresh sapwood every 10 to 15 days; thus fresh sap-oozing wounds were maintained on these trees.

A study, experiment IV, was started at Lehigh, Iowa on May 11, 1954, in which four red oak trees were wounded around an inoculum-bearing tree. Three of these were approximately 100 feet from the source tree and the other one was about 300 feet away. Four red oak trees were also marked as checks. They were from 100 to 150 feet from the mat- and pad-bearing tree.

On May 21, 1954 at Dolliver Memorial State Park in experiment V, four red oaks which were situated in a ring approximately 100 feet from a single mat- and pad-bearing oak in an otherwise oak wilt-free area were wounded and the nearest red oak to each of these trees was marked as a check. During the same day, experiment VI was established in the same park. A lone red oak which was 200 feet from the nearest mat- and pad-bearing tree was wounded, and four nearby red oaks were marked as checks; one located in each direction from the wounded tree.

Results

The bar-graph (Figure 1) shows the number of trees per ten-tree plot that had wilted as of leaf-fall, 1954, in experiment I. The highest percentage of infection occurred in those trees wounded on May 11 and 21, 1954. Four of the ten trees wounded on May 11 died, and three of the ten trees wounded on May 21 died. The earliest infection occurred in plot II of April 30 and the last trees to wilt were wounded on June 22. Of 100 check trees which were located at random within the study area, one died of oak wilt. This tree had the main limb in its crown completely girdled for approximately 3 feet by rodent gnawing during the spring and the tree developed progressive wilt symptoms from this wound point and died.

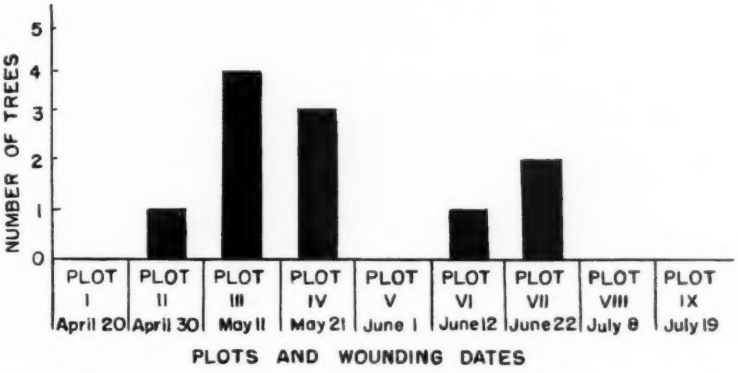

FIGURE 1. NUMBER OF TREES THAT WILTED IN EACH PLOT OF TEN RED OAKS IN EXPERIMENT ONE.

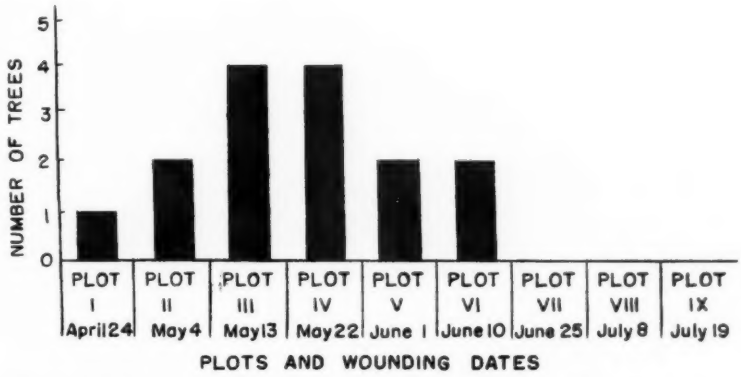

FIGURE 2. NUMBER OF TREES THAT WILTED IN EACH PLOT OF EIGHT RED OAKS IN EXPERIMENT TWO.

In experiment II (Fig. 2), the highest percentage of trees wilted in the plots which were wounded on May 13 and 22. One tree in the earliest wound plot (April 24) died of oak wilt, and the last trees to die were wounded on June 10. Out of 100 check oaks, one died of oak wilt. This tree was wounded by rodents during the spring and the wilt symptoms developed first in the region of the gnawed area.

One of the five trees bearing wounds which were extended into fresh sap-wood every 10 to 15 days wilted in experiment III. None of the check trees wilted.

None of the trees involved in experiment IV wilted.

Of the four red oaks wounded in experiment V, one tree was wilting on July 10, and two more were completely wilted on September 2; none of the checks wilted. In experiment VI involving one wounded tree and four checks, the wounded tree was wilting on July 10, and none of the checks wilted.

Discussion

At the time the first of this series of experiments was originated, the apparent importance of a pre-existent wound in an oak in order for that tree to be infected by insect-transmitted oak wilt fungus had been demonstrated (2, 3, 4, and 7). With one exception (5), all subsequently reported research has strongly substantiated this idea. This lone exception involved the experimental transmission of the oak wilt fungus by the bark beetle species, Pseudopityophthorus pruinosus (Erichh.), to two oak seedlings. The involved beetles were placed in Petri dish cultures of Endoconidiophora fagacearum for 1/2 hour prior to caging on test trees. In order for this species of insect to be accepted as a possible vector of the oak wilt fungus in nature, scientific evidence that this beetle practices habits in nature that afford it access to such an inoculum source must be produced.

Extensive observations throughout the oak wilt-infected areas of Iowa during 1954 failed to yield a single case of isolated-tree infection in which recent wounds were not present on the tree. This does not prove that such wounds were always the entrance for infection, but the 100-percent association of wounds with isolated infections strongly substantiates the wound theory.

The experimental results reported in this paper appear to further support the role of pre-existent wounds in "long distance" transmission of oak wilt. Of 122 red oaks wounded from April 24 to June 22 inclusive, 31 trees died of oak wilt. Of 217 red oak checks, two trees wilted. Both of these trees were wounded by rodent gnawing, and the wilt symptoms developed first in the regions distal or adjacent to the wound area. The vectors in these two cases were apparently the wounding rodents or the insects attracted to the wounds. Many insects were frequently observed on such rodent-inflicted wounds on oaks during this period.

In these tree-wounding studies, which have been continued to the present time, the trees that have wilted as of leaf-fall, 1954, are among those wounded during the interval from April 24 to June inclusive. Some of the trees wounded during the fall period of mat and pad production may wilt during the coming spring and summer of 1955, so no conclusions about the overall yearly periods of natural overland spread can be made, but the data (Figures 1 and 2) indicate that the period of spring and early summer overland dissemination in Iowa is apparently limited to an interval extending from late April until late June. This period corresponds to the spring and early summer period of mat and pad production in Iowa. The data also show that in both major experimental areas the largest percentage of wilted trees occurred in those wound plots which were established during the period from May 11 to 22 inclusive. The results of experiment VI show that three or four wounds on a red oak in the midst of a group of oak wilt-free red oaks can attract vectors carrying sufficient inoculum to infect and kill the tree. The findings of the several other reported small studies also prove that natural overland transmission of Endoconidiophora fagacearum to wounds on red oak trees does occur in Iowa when the inoculum and the suitable wound are provided and the other necessary environmental conditions exist. The prevalence with which such dissemination occurs in Iowa during the previously discussed conditions and period is shown by the fact that overland spread occurred in five out of six separate experimental areas located in two different State parks.

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OCCURRENCE OF OAK WILT FUNGOUS MATS AND PADS ON MEMBERS OF THE RED AND WHITE OAK GROUPS IN IOWA[1]

Arthur W. Engelhard

Abstract

Oak wilt fungous mats and pads have been observed on members of both the red and white oak groups in Iowa. Mats and pads on members of the red oak group were first observed in July 1952, and on bur oak, Q. macrocarpa Michx., a member of the white oak group, on May 11, 1954. The fungous structures have been observed during all twelve months of the year. Between 1952 and 1954 they were found to be formed most prolifically during two periods: The first beginning the third week in August and continuing through October, and the second beginning the third week in April and continuing through the first week in June.

Introduction

Fungous mats and pads on trees of the red oak group killed by Endoconidiophora fagacearum Bretz have been generally observed in all sections of eastern United States where oak wilt is known (1, 2, 3, 5, 6). In Iowa they were first observed on members of the red oak group in July, 1952, and on bur oak, Quercus macrocarpa Michx., a member of the white oak group, on May 11, 1954. In order to determine the time, frequency and abundance of mat and pad formation, wilt-killed trees were observed over a 2 year period in Pilot Knob State Park, Forest City, Iowa.

Occurrence of Fungous Mats and Pads on Trees of the Red Oak Group

Although mats and pads have been seen in Iowa during every month of the year, observations from the fall of 1952 through 1954 revealed two periods of prolific mat and pad growth during the year. Formation began on recently wilted oak trees during the last week in August and continued through October, when, apparently, the onset of lower temperatures was instrumental in gradually slowing down and bringing the active fall mat- and pad-forming period to a close. In the spring they were again prolifically formed beginning the third week in April and extending through the first week in June. They were formed only on trees that had died the previous season, and it was common for the same tree to have new mats formed in both the fall and spring periods.

Between the fall and spring periods, mats and pads in good physical condition were commonly observed. During December, 1953, wood and bark sections containing mats and pads were removed from two wilt-killed red oaks. The wood and fungous structures were frozen solid, but, upon thawing, the mats and pads had a very fresh appearance as if they had recently been produced. Similarly in March, 1954, mats and pads just beginning to age (darken in color) were found, as well as perithecia with extruded "ascospore-droplets." Freshly produced perithecia were common in both the fall and spring on the mats.

Although the two major mat- and pad-forming periods on trees killed by E. fagacearum are in the fall and spring, the frequent occurrence of fresh appearing mats and pads during the winter months can be attributed to their being produced possibly late in the fall and then being frozen in situ throughout the winter.

Very few mats and pads were observed in the period beginning the second week in June and extending through the second week in August.

Tremendous variation existed among red oaks in the number of mats and pads formed per tree. In some instances trees were found that apparently formed no mats and pads, whereas one northern pin oak, Quercus ellipsoidalis Hill, 12 inches d.b.h. and 40 feet tall, was found to have at least 246 of the structures scattered from the base of the bole to points high in the

[1] Journal Paper No. J-2699 of the Agricultural Experiment Station, Ames, Iowa. Project No. 1047. Research sponsored jointly by the National Oak Wilt Research Committee, the Iowa State Conservation Commission and the United States Department of Agriculture.

crown on branches as small as 1 inch in diameter. Of these 246 fungous structures, 84 had been gnawed upon by rodents. Twenty-four additional gnawed areas were found on small branches but it was difficult to ascertain at the time of inspection if fungous mats and pads had been present. Two other red oaks, 11 inches and 12 inches d.b.h. standing nearby, were debarked in the lower 7 feet of the bole and were found to contain 65 and 75 fungous mats and pads, respectively. Although the number of mats and pads present on these trees is believed to be above average, it does point out how prevalent these structures may be on members of the red oak group, and suggests that abundant inoculum may exist at certain times in oak wilt areas.

Occurrence of Mats and Pads on Trees of the White Oak Group

According to the most recent information available, oak wilt fungous mats and pads have not been hitherto reported or seen on any member of the white oak group.

On May 25, 1953, a bur oak, 8 inches d.b.h. and 46 feet tall, and nine trees of the red oak group were inoculated with fresh E. fagacearum "ascospore-droplets." Eight of the nine red oaks and the bur oak developed typical oak wilt symptoms in the tops of the crowns within 3 weeks. In all of the wilting trees the symptoms spread quickly throughout the crowns and defoliation was nearly complete by the end of the summer. The bur oak produced a few water sprouts late in the summer and was not observed to produce any fungous mats and pads during the fall; admittedly, they could easily have been overlooked.

On May 11 1954, it was noticed that rodent-gnawing had occurred on the bur oak, similar to that seen many times on mat- and pad-bearing red oaks in the area. Closer examination revealed that this bur oak was producing fungous mats and pads similar in external structure, appearance, and fruity odor to those produced on members of the red oak group. The tree was examined still more closely and was found to bear 42 of the fungous structures. Not all contained both a mat and a pad, for a number of the smaller ones were composed of only a pad-like portion which also is common on red oaks. Rodents had gnawed on fourteen of the mats and pads.

Discussion

It is apparent that fungous mats and pads are very common and can be found easily on members of the red oak group. On members of the white oak group, however, they are apparently very uncommon and also somewhat difficult to locate owing to the furrowed, ridged, or flaky structure of the bark. It was found also that the usually successful method of locating mats and pads on red oaks, i.e., tapping the bark for hollow spots with a plastic mallet or thick elongate bottle, was not so successful on bur oaks because the overlapping, ridged bark had a tendency to sound hollow even on living, healthy trees. Thus, there is a possibility that mats and pads on members of the white oak group may not be quite as uncommon as previously suspected, for they may have just been generally overlooked.

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LONGEVITY OF THE OAK WILT FUNGUS IN OAK ROOTS AS RELATED TO SPREAD THROUGH ROOT GRAFTS

William L. Yount

The oak wilt organism Endoconidiophora fagacearum was first reported isolated from all parts of infected trees including roots, stems, branches, twigs, and leaves, by Henry in 1944 (2). Kuntz and Riker (3) in 1950 pointed out the significance of preexistent natural root grafts between healthy and infected oaks as a means of local spread of the oak wilt disease. Workers in several States have reported longevity of the fungus in roots: Ohio, 9 months (5); Pennsylvania, 1 year (4); Wisconsin, 3 years (1). It has been suggested (1) that the long life of some diseased roots is due to the established grafts with healthy oaks that support and nourish them.

Present Work and Observations

An uncompleted study of the time intervals between deaths of trees within the same area where the distances between trees suggest infection by root grafts, shows lapses, in many cases of 2 or more years. Several areas treated in 1951 (cutting trees in a 50-foot area around infected trees, and treating with Ammate) have wilted trees on their perimeters in the 1954 season. If the infections were due to root grafting, the intervals between tree deaths may be explained on the basis that the wilt organism lives for some time in diseased roots that are already or subsequently become grafted to healthy trees.

In an attempt to discover how long Endoconidiophora fagacearum lives in the roots of Pennsylvania oaks, isolations were made from trees of known histories. Trees with well preserved roots were selected. Preliminary attempts to isolate from partially rotted roots were negative. Results are shown in Table 1.

Table 1. Isolations from roots of oak wilt trees.

Year tree died	Number of trees checked	Number of positive isolations
1950	5	0
1951	3	1
1952	8	5
1953	7	5

It was found that the oak wilt organism can live 3 years or more in diseased roots of Pennsylvania oaks.

Discussion

The established eradication practice is to fell the diseased trees and all healthy trees within 50 feet. The stumps are treated with Ammate. Recent isolations from diseased trees so treated have shown that Ammate does not kill the fungus in the roots. Such roots if connected with the roots of surrounding stumps and these in turn with roots of healthy oaks on the perimeter of the treated area constitute sources and avenues of inoculation and subsequent spread of the disease. Yearly inspection of such treated areas adds to the expense of an eradication program.

A chemical treatment for the stumps to impregnate all roots with a poison that will prevent growth and passage of the fungus through the roots is needed. An area so treated would insure against further new infection resulting from diseased roots in the soil.

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CONTROL OF MAT FORMATION BY THE OAK WILT FUNGUS BY EARLY FELLING OF DISEASED TREES

C. L. Morris[1]

Summary

A total of 182 trees in varying stages of foliar wilt resulting from infection by the oak wilt fungus were treated during the summer months of 1954 in an effort to reduce the number of mycelial mats formed on them. Three methods were used: (a) felling, (b) deep girdling, and (c) the application of sodium arsenite painted on a band of exposed sapwood around the trunk.

Mycelial mats were produced on only 1.3 percent of 153 trees treated in stages of defoliation ranging from 20 to 100 percent by the three methods. All trees in this group which were wilted completely were treated within two weeks of the time defoliation was complete. In comparison, mats were produced on one-third of the 29 infected trees which remained standing six weeks or longer before treatment.

One of the major objectives following the discovery of oak wilt (Endoconidiophora fagacearum) in Pennsylvania in 1950 has been in the development of efficient, simplified and economical techniques of field control. This paper reports the results of one phase of this objective; i. e., the practicability of felling diseased trees in various stages of foliar wilt to reduce or eliminate mat production. If practical, early felling would reduce labor requirements to a fraction of that needed for the wholesale peeling of diseased trees, a previously accepted control measure in Pennsylvania.

Boyce (1) recently published results of 2-year study of mat formation by the oak wilt fungus on felled versus standing trees with the idea of forcing mat production on infected trees during the late summer season. His data showed that mats formed naturally on at least half of the standing untreated trees, whereas mats were produced on only 11 percent of the felled trees.

Struckmeyer and Kuntz (4) in their histological studies pointed out the rarity of mycelia of the wilt fungus in the vessels of infected northern pin oaks at the time of incipient wilt, later noting extensive growth of mycelia in the vascular elements as severe wilt symptoms and defoliation developed. Similarly, Young (5) noted that the fungus was often well distributed in longitudinal streaks in the boles of wilting trees, but not completely around the circumference. In another study, Boyce (2) attempted a total of 727 isolations, 310 from the crowns of wilting red oaks and 417 from the boles of the same trees. Forty-five percent of the crown isolations were positive; only 27 percent of the isolations from the bole were positive. These investigations point to a "spotty" distribution of the fungus in the bole and many of the larger branches during the early stages of wilting. This fact is probably mainly responsible for the absence of mats on most diseased oaks treated before foliar wilting is complete.

Experimental treatment of standing diseased red oaks with solutions of copper sulfate in Pennsylvania often shows appreciable upward movement of the solution in the outer layer of sapwood of the bole even after the tree has been completely defoliated for a period of several weeks. This continued translocation of fluids in vascular tissues, which may remain alive for a period of weeks after wilting, is apparently important in the further distribution of the fungus in the tree and subsequent mat formation.

The first indication in Pennsylvania that early felling of diseased trees would eliminate mat production was reported by Morris and Fergus (3). In this case, one of two infected companion sprouts in the same stage of foliar wilt was felled in late August. While no mats were formed on the felled stem, a large number were noted the following spring on the other standing sprout. Ten additional trees, all approximately 50 percent wilted, were felled in the autumn of 1952 and 1953. No mats were produced on any of these trees.

To test the validity of these early observations a total of 172 diseased trees in various stages of foliar wilt were treated by several different methods during the growing season of 1954. The majority were felled or girdled completely through the sapwood. The remainder were treated by painting sodium arsenite on a band of exposed sapwood around the trunk. These trees were scattered throughout several counties in southcentral Pennsylvania.

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[1] Credit for most of the necessary field work goes to Ralph Sanderson and H. E. Thompson of the Pennsylvania Department of Agriculture. Technical advice and assistance by F. C. Craighead, consulting entomologist, Pennsylvania Department of Agriculture, is gratefully acknowledged.

Table 1. Summary of data on early felling of diseased trees to control mat production.

No. of trees treated in months indicated - 1954		Method of treatment	Average defoliation at time of treatment (% foliage lost)	No. trees 100% defoliated when treated	Trees on which mats produced (%)
June-July	Aug.-Sept.-Oct.				
63	26	Felled	70	5	1.4
0	29	Felled	100[a]	29	31.0
50	4	Deep girdle[b]	55	7	0.0
10	0	Chemical debarking	45	0	0.0
(9) check trees allowed to stand		None	-	-	11.0

[a] Trees completely defoliated 6 weeks or longer before treatment.

[b] All girdles treated with dry crystals of copper sulfate, Ammate, or combination of these two chemicals.

Seventy-five percent of the treated trees apparently were infected through root grafts; the remainder were inoculated. Table 1 summarizes the results of various treatments.

Of the three variations in control methods applied in this experiment, painting the cambial area with a solution of sodium arsenite -- a method now used extensively for "chemical peeling" of pulpwood -- resulted in most rapid kill of the cambium and subsequent drying of the bark and outer sapwood of the boles and tops. However, this method was applied to only a small number of infected trees which had lost less than 60 percent of their foliage. Felling the diseased trees or allowing them to remain standing after applying a deep girdle completely through the sapwood was also quite effective with only two exceptions noted. Mats were produced in September on one 6-inch red oak, 50 percent defoliated when felled July 9, and in October on one 15-inch red oak, 85 percent defoliated when felled July 29.

Although those trees which were girdled and allowed to remain standing received additional treatment in the form of dry crystals of copper sulfate, Ammate, or combinations of these two chemicals to hasten root kill, it is felt that deep girdling without the addition of any poison would have been as effective in reducing mat production.

Of a total of 29 trees which were 100 percent defoliated but which had remained standing for 6 weeks or longer before treatment, nine trees had mats produced on them by December 1954.

Nine infected trees which showed first wilt symptoms in late June, in July, or August, were allowed to remain standing as checks until December 1954, when an examination showed only one had reached a condition where mats would ordinarily be formed. A few mats had matured on this tree earlier in 1954. The remaining check trees were still green.

Mr. Arthur Jeffery, Pennsylvania Department of Agriculture, has tested the application of the early felling technique in the routine field control measures for oak wilt in southcentral Pennsylvania during 1954. He reports that a total of 811 trees, the majority in the early stages of foliar wilt, were felled during late June, July, August, and early September. These trees were examined in October and November 1954. One hundred fifty-three were still green and were peeled at the time of examination. Of the remaining 658, only nine (all felled during August and September) showed evidence of mat formation.

Precipitation in central and southcentral Pennsylvania, where the experimental and routine field control measures were applied, was sub-normal, although not droughty, in July and August of 1954. Since relatively few "check" trees were included with the experimental treatments, the full effect of dry weather in reducing mat formation on diseased trees cannot be fully evaluated at the present time. In view of the number of trees treated and the results to date, however, dry weather had little or no effect in reducing mat production in Pennsylvania during 1954.

Severing the vascular elements of diseased trees by felling or by deep girdling allows additional treatment of the root system by poisoning. Application of sodium arsenite to the exposed sapwood holds particular promise as an effective treatment, at least for partially wilted trees, if treatment of the root system of the diseased tree is deemed unnecessary.

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A SOUTHWIDE SURVEY FOR SWEETGUM BLIGHT

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Sweetgum (Liquidambar styraciflua L.) has been showing signs of decline to varying degrees over much of its range in recent years. Dieback involving part or all of the crown, sometimes leading to the death of trees, has been observed for many years. More recently an accentuated decline, reaching alarming proportions in parts of several States, particularly in eastern Maryland, has been receiving research attention.

The symptoms of a sweetgum leader dieback have been described by Garren (2) and those of what is called sweetgum blight by Miller and Gravatt (3), Toole (4), and Young, Toole and Berry (5). The cause of neither decline is known, and there is as yet no proof that they are different diseases. While the symptoms are different in some respects, there are enough similarities to make distinction between them impossible in many areas. Therefore, none was made for purposes of a 1954 survey to determine the condition of sweetgum over the South in general. Thus the term sweetgum blight is used here to refer to the entire complex of crown deteriorations.

Earlier surveys (5) had shown blight symptoms to be prevalent from Maryland to Texas, but data were not provided in terms of numbers of trees, volume of affected timber, or the condition of sweetgum as compared with associated species. In 1954 a survey was designed to provide such data, and Fields W. Cobb, Jr., and Robert Wagner, on temporary assignment with the Southeastern Forest Experiment Station, carried out the field work between June 15 and August 31[2].

Methods of Sampling

Detailed data on sweetgum and associated species were obtained on four strip plots, established at right angles to roads, in two upland and two coastal counties, wherever possible, in each Atlantic and Gulf Coast State from Delaware to Louisiana. The counties were selected as being representative for sweetgum. The plots, taken at random, were 1/2-chain wide, and as long as necessary to tally at least 30 sweetgums 5 inches in d.b.h. or over. The diameters of these trees and all associated species on the plots were measured and the extent of dying of the crown of each tree recorded. In four States the full 16 plots were not obtained, but in Maryland, owing largely to supplementary surveys by State crews, 67 plots were obtained. The ten Delaware plots were also measured by State personnel.

While the strip plots were useful for species comparisons and board-foot volumes, they still represented information on only 190 plots over ten States. Thus a second type of plot was taken, roughly every 5 miles of road over the entire summer's travel route, if a stand of sweetgum averaging 5 inches or more d.b.h., and large enough for a 1/4-acre circular plot, could be conveniently sampled. On these circular plots no trees were tallied, but each plot as a whole was designated in accordance with the classifications given in Table 1. The data obtained on 1,298 such plots, together with the more detailed data on the 190 strip plots, give a fair appraisal of the condition of sweetgum in the States surveyed. No circular plots were taken by the State crews in Delaware and Maryland.

Results

The strip plot data show that on a Southwide basis sweetgum exhibited more crown deterioration than any other species, with one-third of the 4,934 trees tallied showing some dieback (Table 2). The elms were almost as bad. In all categories of crown blight or dieback, sweetgum stands out. However, in terms of percentage of trees dead, sweetgum was no different from most other species, even in Maryland.

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[2] The Southern Forest Experiment Station Forest Survey and Pathology staffs and the Southeastern Station Forest Survey staff assisted materially in planning the sweetgum survey. The Northeastern Station also cooperated and enlisted Delaware and Maryland assistance. The plots taken by crews provided by these two States greatly strengthened the data for the middle-Atlantic area.

Table 1. Sweetgum blight incidence as estimated on 1,298 1/4-acre circular roadside plots surveyed in 9 States.

Amount of blight	States surveyed									Total plots	
	Md.	Va.	N.C.	S.C.	Ga.	Fla.	Ala.	Miss.	La.		
	Percentage of plots									No.	%
Incidence of blight on plots											
No blight	12	40	42	34	38	50	20	22	33	424	33
1-25% trees affected	15	38	38	37	29	29	38	43	44	466	36
26-50%	33	18	16	23	26	10	31	28	15	291	22
Over 50%	40	3	4	7	6	10	11	7	7	117	9
Prevailing extent of dieback on plot trees											
None	12	40	42	34	38	50	20	22	34	424	33
1-20% dieback	67	34	32	25	24	20	24	38	41	406	31
21-50%	21	23	19	30	29	20	48	37	21	384	30
Over 50%	0	2	7	11	9	9	8	3	4	84	6
Number of plots	66	146	144	142	170	133	209	174	113	1298	

Table 2. Condition of crowns of all trees on strip plots, 5 inches d.b.h. and larger, all States. (Intermediate and suppressed trees excluded).

Species	States surveyed									Total trees examined	
	Del.	Md.	Va.	N.C.	S.C.	Ga.	Fla.	Ala.	Miss.	La.		
	Percentage of total trees healthy										No.	Percent
Sweetgum	80	50	77	72	69	73	69	59	71	53	3144	64
Black or (tupelo gum)	90	94	80	87	82	90	94	86	87	95	484	87
White oak	96	93	85	77	86	78	89	98	87	85	504	88
Red oak	94	93	74	85	89	85	87	92	93	90	884	90
Maple	92	94	68	80	72	88	100	79	67	80	457	83
Yellow-poplar	100	93	-	90	90	89	83	97	88	-	287	93
Elm	-	-	63	64	78	70	75	63	68	-	79	68
Miscellaneous species	100	90	85	68	75	91	93	87	80	82	793	86
	Percentage of trees with up to 1/8th of crown dead or dying											
Sweetgum	14	22	13	21	24	19	17	25	16	32	1017	21
Black or tupelo gum	10	6	15	9	18	7	5	10	4	0	55	10
White oak	2	4	10	19	9	11	11	2	9	15	50	9
Red oak	4	6	13	8	11	5	7	4	4	10	60	6
Maple	3	6	28	13	17	6	0	13	25	10	66	12
Yellow-poplar	0	6	-	10	4	11	0	3	0	-	16	5
Elm	-	-	25	16	7	20	25	13	21	-	19	16
Miscellaneous species	0	9	12	17	18	5	3	8	14	4	82	9
	Percentage of trees with more than 1/8th of crown dead or dying											
Sweetgum	5	27	10	6	5	8	12	12	11	11	697	14
Black or tupelo gum	0	0	4	4	0	0	1	2	4	5	11	2
White oak	0	0	4	4	5	11	0	0	0	0	16	3
Red oak	0	0	10	5	0	8	4	4	3	0	31	4
Maple	5	0	5	6	7	6	0	5	8	10	23	5
Yellow-poplar	0	0	-	0	5	0	0	0	0	-	4	2
Elm	-	-	12	16	14	10	0	19	8	-	15	13
Miscellaneous species	0	0	3	11	8	2	4	3	4	8	32	4
	Percentage of trees dead, not due to injuries											
Sweetgum	1	1	0	0	1	0	1	4	2	5	74	1
Black or tupelo gum	0	0	1	0	0	3	0	1	4	0	4	1
White gum	2	2	1	0	0	0	0	0	4	0	5	1
Red oak	2	2	3	2	0	1	1	1	0	0	9	1
Maple	0	0	0	1	2	0	0	3	0	0	4	1
Yellow-poplar	0	0	-	0	0	0	17	0	12	-	2	1
Elm	-	-	0	1	0	0	0	6	3	-	33	3
Miscellaneous species	0	0	1	3	1	1	1	1	2	6	10	1

Table 3. Volume and number of sweetgums affected by blight, on 190 strip plots.

Tree condition	States surveyed										Total volume	
	Del.	Md.	Va.	N.C.	S.C.	Ga.	Fla.	Ala.	Miss.	La.		
	Percentage of total board-foot volume										(1000 board feet)	%
In healthy trees	85	46	92	70	57	61	63	56	68	52	188.1	60
Trees with up to 1/8 crown dead	12	20	6	25	36	28	24	30	21	37	80.5	26
Trees with 1/8 to 1/2 crown dead	2	27	1	4	4	7	11	9	8	7	33.7	11
Trees with crowns over 1/2 dead	1	1	1	1	1	3	1	2	1	1	7.4	2
Dead trees	0	0	0	0	2	0	1	4	2	3	4.7	1
Total volume (1000 board feet)	11.1	58.7	10.5	17.3	31.6	33.7	31.6	42.5	45.8	31.6	314.4	
	Percentage of total trees										Total trees	
											No.	%
Healthy trees	82	50	77	72	68	73	70	59	71	53	3144	64
Trees with up to 1/8 crown dead	14	21	13	21	24	19	17	25	16	32	1015	20
Trees with 1/8 to 1/2 crown dead	3	20	6	4	6	7	11	9	9	8	546	11
Trees with crowns over 1/2 dead	1	6	4	2	1	1	1	3	2	2	156	3
Dead trees	0	1	0	1	1	0	1	4	2	5	73	1
Total trees	286	1426	256	331	480	477	479	479	480	240	4934	
Number of strip plots	10	67	12	13	16	16	16	16	16	8		

In terms of sweetgum board-foot volume (in trees 11 inches d.b.h. or larger), 60 percent of the volume was in healthy trees, 26 percent in trees with up to 1/8 of the crown dead, 11 percent in trees with between 1/8 and 1/2 of the crown dead, 2 percent in worse condition, and 1 percent in dead trees (Table 3).

On a State basis sweetgum was in the poorest condition in Maryland, although blight was severe locally in several other States, particularly in South Carolina, Florida, Alabama, Mississippi, and Louisiana.

The data on the 1,298 circular plots on which data were taken only on the sweetgum also show crown-dieback to be common, with only one-third of the plots without any noticeable crown blight. Again, as in the case of the strip plots, most of the trees had less than 20 percent of their crowns dead. Maryland had the fewest entirely healthy plots, and the area from South Carolina to Alabama had the most plots in which the prevailing dieback observed involved 50 percent or more of the individual crowns affected (Table 1).

Sweetgum blight was twice as common rolling or upland sites as on flat or bottomland sites, but occurred on a wide variety of soil types and drainage situations.

The Federal field crew made an effort to record general observations on the two prevalent types of blighting. The "leader dieback" is typified by a non-progressive stagheading, with a sharp line of separation between the dead top and healthy part of the crown. It is commonest in older trees. The "sweetgum blight" is often progressive with intermediate zones of dwarfed or discolored foliage between the dead and normal parts of the crown, is common in trees of any size, and is often fatal. Borderline cases between the two conditions often precluded easy separation, hence the plot records made no distinction. Notes by Crew Chief Cobb on the two types, by States, are as follows:

Maryland -- Blight particularly prevalent, overshadowing possible dieback.
Virginia -- Blight common in northern counties but less so than in Maryland. Dieback clearly apparent in southeastern Virginia.
North Carolina -- Blight mostly in Piedmont. Dieback common in extreme eastern counties.
South Carolina -- Blight common in all areas sampled. Dieback also scattered in both Piedmont and Coastal Plain.
Georgia -- Blight evenly distributed over the State Dieback occasional but most common in southern part of State
Florida -- Eastern part had little blight, but west of Tallahassee it was common. Dieback common in Florida.
Alabama, Mississippi, and Louisiana -- Blight severe over these States but worst in the southern parts. Dieback also common.

Discussion

Throughout the South, although sweetgum blight is common, it has not caused excessive mortality. Only 1 percent of all sweetgums tallied were dead, and only 1 percent of the Maryland sweetgums were dead. The mortality of all species other than elm was also 1 percent. Three percent of the elms were dead (Table 2).

On a sawlog volume basis, 86 percent of the board-foot volume of sweetgum was in trees healthy or with up to only 1/8 of the crown dead. Although this was the average situation, there have been serious declines of sweetgum in Maryland and locally in parts of most other southern States.

It is understandable that there should be some alarm at the rate at which second-growth sweetgum has been dying around College Park, Maryland, Stoneville, Mississippi, Gainesville, Florida, Middlesboro, Kentucky, and many other widely separated areas. With the cause of the so-called sweetgum blight and related or similar gum decline still unknown, such epidemic mortality is reason for apprehension even if localized. The average condition of sweetgum, however, as determined from the 1954 survey, indicated no serious general mortality, but rather a tendency to limited crown deterioration. While this latter situation is particularly aggravated in Maryland, perusal of the attached tables shows several States with incidence and severity of crown blighting closely paralleling that in Maryland.

In every State in which species of elm were tallied, their condition was as bad as sweetgum; and in terms of severe dieback and death, the elms led sweetgum. Some other associated species, particularly maples and sometimes oak, sycamore, and ash, were in a condition similar to sweetgum in several stands.

Young (6) finds that sweetgum plots in Maryland are declining materially from year to year, and Toole (4) finds a similar situation on some Mississippi plots, with little change in others, and, while finding conspicuous fine-root mortality on blighted trees, has no evidence on the cause. Garren (2), Miller and Gravatt (3), and Berry (1) have also sought the cause but have yet no promising leads. Thus we can say that we have one or more sweetgum blights, and that they or it causes real damage in localized areas in almost every State in the sweetgum range, that mortality has not been excessive except very locally, that the blight condition is currently continuing. We must also add that its cause is not known.

The situation at present suggests maximum utilization of sweetgum wherever the blight is severe or increasing, and the replacement of sweetgum shade trees in the many urban and suburban areas where important losses have been occurring. In the absence of knowledge on the cause or on foci of spread that would indicate a pathogen, it would appear still unjustified or at least premature to take a position that sweetgum is on its way out because of disease over any major part of its range. The Southwide distribution of sweetgum crown diebacks suggests that climatic or soil influences may have a role in these disturbances, although the Maryland developments may indicate a distinct and particularly aggressive type of decline.

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PROGRESS OF SWEETGUM BLIGHT IN MARYLAND PLOTS, 1952-1954

George Y. Young

The first report of the sweetgum blight (cause undetermined) in Maryland was in Prince Georges County; its later rapid increase in this area has been described elsewhere (2, 3, 4). Hundreds of dead or unsightly shade trees have been cut down in University Park and neighboring College Park.

A description of the symptoms is given by Miller and Gravatt (3). One item here is worthy of comment. The premature discoloration of the foliage they describe appears to have diagnostic value even when limited to single branches. During late August and early September 1953, 18 such cases of single-branch discoloration were tagged for observation. In 15 of the 18 cases the branches leafed out in the spring of 1954 but the foliage, although green, never developed to normal size. During the spring and summer of 1954, many of these branches died in typical blight fashion.

The observations on Maryland progress plots previously reported (4) were continued for another year, as well as on new plots established in 1953. All were within 60 miles of Beltsville and most of them closer. The results for the entire period are shown in Tables 1 and 2. The percentage of healthy trees decreased in every plot, sharply in most of them. Five of the plots contained dead trees when established, and dead trees appeared in all of them during the period of observation. Figure 1 indicates that the deterioration was nearly as rapid from September to May as in the summer months, and continued at an approximately uniform rate throughout the period of observations. In Figure 1, the graph for the 1951 plots showed a slight tendency to flatten out in 1954, but flattening in this part of the percentage range is in accordance with normal probability and does not indicate any diminution in the activity of the causal factor or factors.

In each examination, trees were rated on the following classification, based on twigs or branches that were dead or defoliated. Trees showing only leaf dwarfing or discoloration were counted as healthy.

Class 0: Apparently healthy
1: Up to 1/8 of the crown affected
2: 1/8 to 1/2 of the crown affected
3: 1/2 or more of the crown affected
D: Dead tree

Occasional trees recorded in Class 1 have appeared healthy at the next examination, probably because the killed twigs were broken and blown off by winter winds and snows; or because winter-killed twigs were mistaken for blight at an earlier examination. In no case in the plot studies has a tree in Class 3 shown subsequent improvement. In September 1954, combining all plots, 28 percent of the trees were healthy, 25 percent in Class 1, 17 percent in Class 2, 15 percent in Class 3, and 15 percent dead.

These progess plots were not selected as indicating the frequency of the disease in the State or even this portion of it. A survey for that purpose will be reported by the Southeastern Forest Experiment Station. More than half of the trees were growing under shade tree conditions in the towns of College Park, University Park and Greenbelt, Maryland. The 1952 plots showed approximately half, and the 1953 plots nearly a third, of the trees already diseased when the plots were selected, and were thus very likely worse than the average stands of sweetgum in parts of the State with which the writer is familiar.

In August 1952 a study of the effect of fertilization on the progress of sweetgum blight in individual ornamental trees was initiated at Greenbelt, Maryland, and the National Arboretum in Washington, D. C. The fertilizer was applied in crowbar holes 12 to 14 inches deep and about 2 feet apart. At Greenbelt the rate of application was 2 lbs. of 10-6-4 fertilizer per inch of diameter at breast height. At the Arboretum the rate was 3 lbs. of nitrogen (18.75 lbs. of 16% nitrate of soda) per tree regardless of size. Treatment in both areas was repeated in April 1953.

The condition at the time of the first treatment, size, and number of trees fertilized in this study are given in Table 3.

On three circular 1/10-acre forest plots (Plots 1, 8, and 9 of Tables 1 and 2), fertilizer was broadcast on the surface of the ground in April 1953. Application was at the rate of 2,000 lbs. per acre of 10-6-4 fertilizer. Each plot was divided into four sectors and alternate sectors

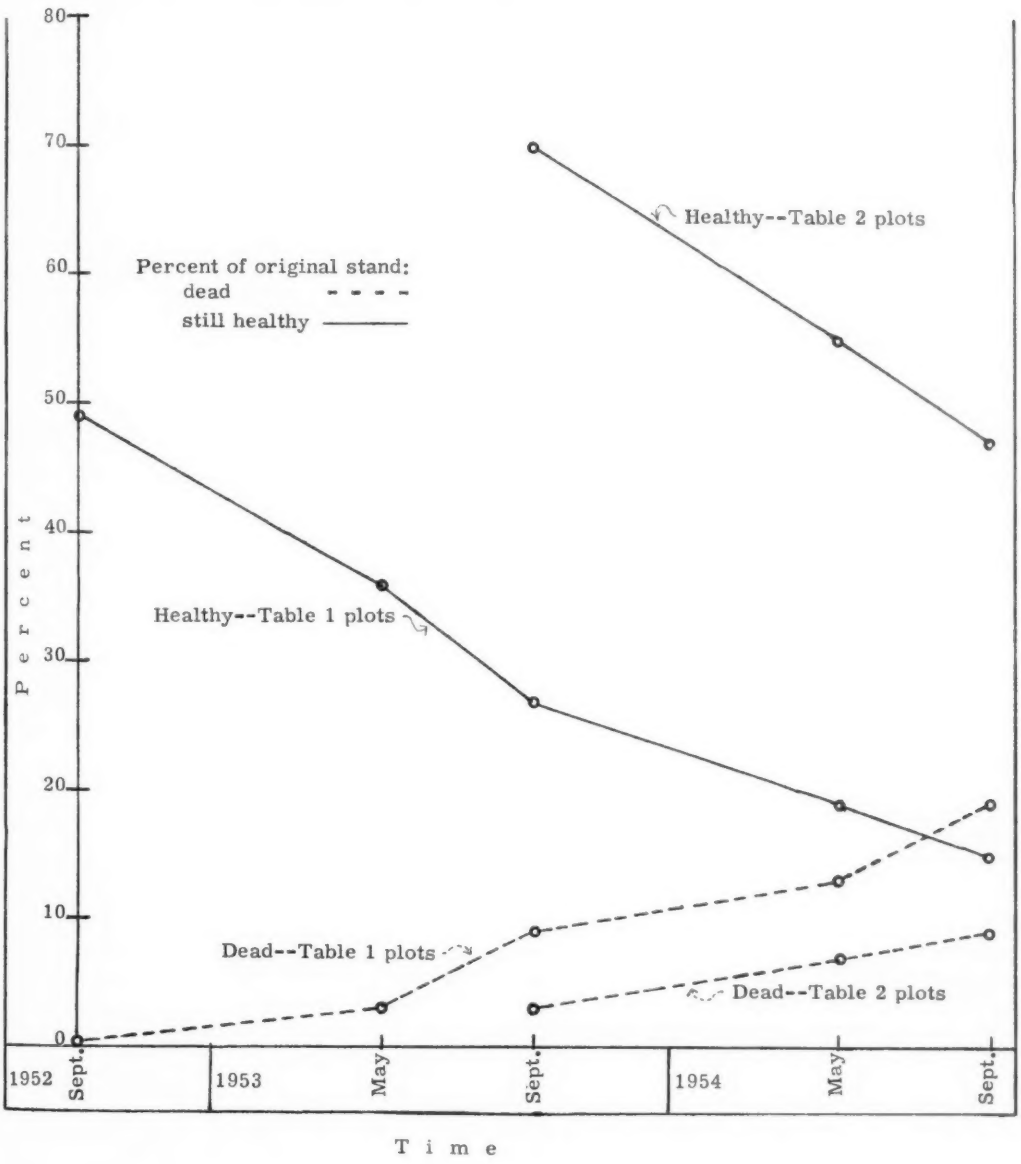

FIGURE 1. Progress of sweetgum blight in Maryland plots, 1952-1954.

Table 1. Original amount of sweetgum blight and incidence in 1954 in plots established in 1952.

Plot number	Number trees in plot	Percent healthy		Percent diseased		Percent dead	
		1952	1954	1952	1954	1952	1954
8	26	73	35	27	61	0	4
24	378	53	14	47	70	0	16
25	238	49	14	51	59	0	27
9	55	46	20	51	64	3	16
19	189	38	14	62	66	0	20
17	15	20	0	80	80	0	20
	901						
Weighted average		49	15	51	66	0.4	19

Table 2. Original amount of sweetgum blight and incidence in 1954 in plots established in 1953.

Plot number	Number trees in plot	Percent healthy		Percent diseased		Percent dead	
		1953	1954	1953	1954	1953	1954
15	47	100	87	0	11	0	2
14	57	93	81	7	14	0	5
12	72	76	40	12	36	12	24
21	44	73	59	27	39	0	2
22	87	69	22	31	73	0	5
10	65	65	54	35	38	0	8
11	73	66	41	34	53	0	6
13	56	61	43	39	50	0	7
1	37	60	54	32	27	8	19
7	47	58	36	38	58	4	6
2	52	46	29	41	50	13	21
	637						
Weighted average		70	47	27	44	3	9

Table 3. Condition, number, and size of fertilized trees.

Tree condition (Class)	Greenbelt		National Arboretum	
	Number trees	DBH (inches)	Number trees	DBH (inches)
0	---		1	8
1	6	4, 4, 5, 5, 7, 10	3	3, 7, 10
2	2	7, 9	5	7, 8, 9, 10, 11
3	3	4, 6, 6	1	6

Table 4. Departures of temperature and precipitation from normal at College Park, Maryland for the periods April through July, 1940-1952 inclusive.

Year	Rain, inches	Temperature °F
1940	- 1.4	- 0.2
1941	+ 2.5	+ 1.8
1942	+ 0.4	+ 2.3
1943	- 3.3	+ 1.9
1944	- 5.1	+ 2.5
1945	+ 10.6	+ 1.0
1946	- 1.3	+ 0.1
1947	+ 5.7	+ 0.7
1948	+ 5.6	+ 0.8
1949	---[a]	+ 1.1
1950	- 0.4	- 0.9
1951	+ 2.6	+ 1.0
1952	+ 5.4	+ 1.4

[a] July record incomplete - very near normal for the period.

were treated.

In April 1953 boron, in the form of borax, was broadcast under five ornamental trees at the rate of 10 lbs. per acre and chelated iron (9%) was broadcast under three additional trees at the rate of 1 1/2 lbs. per tree.

At the end of the 1954 growing season, none of the treatments with fertilizers or trace elements had shown any indication of arresting or controlling sweetgum blight.

Pruning tests in which all diseased parts of a tree were eliminated by cutting off the affected branches well beyond any outwardly visible evidence of the disease were made on ten ornamental trees. Five trees were in Class 1 and five in Class 2. Pruning improved the appearance of the trees for the current season but failed to check the progress of this disease.

Many questions regarding climatic factors have been raised in connection with this disease since it was first reported in 1951 (2). Some pertinent weather data for College Park, Maryland, were compiled, a summary of which is given in Table 4.

These growing season weather records present no abnormality that would seem likely to cause the progressive decline of sweetgum beginning in 1948 or shortly before. For definite conclusions on weather relations a more exhaustive study would be needed.

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FOREST SERVICE, U. S. DEPARTMENT OF AGRICULTURE

INVESTIGATIONS OF POSSIBLE CAUSES OF SWEETGUM BLIGHT

Frederick H. Berry[1]

Sweetgum blight, a disease of unknown origin and cause, was first observed on sweetgum trees (Liquidambar styraciflua) in Prince Georges County, Maryland, in 1948 (2). The disease increased in extent and severity in this section and was described in detail by Miller and Gravatt (3) in 1952 and by Young, Toole, and Berry (4) in 1954. A leader dieback of sweetgum in Alabama, described by Garren (1), is similar in many respects to the disease in Maryland.

Since the papers cited describe in detail symptoms of the disease, distribution and spread, and damage, this information will not be repeated here. This paper summarizes the writer's investigations during 1952-53 on possible causes of the disorder.

Fungi, viruses, nematodes, unfavorable weather conditions, an increase in mean annual temperature over a period of years, unfavorable sites, nutrient deficiencies, and disturbances of soil or ground water have been suggested as possible causes of the disease. Even though sweetgum is a short-lived species in the southern uplands, the increase in incidence of this new sweetgum trouble and the rapid dying of many of the observed trees suggest that the disease may be infectious.

Fungi

One of the first phases investigated was the possibility of the disease being of fungus origin. Miller and O'Brien (2) isolated species of Diplodia, Trichoderma, and Penicillium from various parts of diseased trees.

During 1952 and 1953 the writer made many isolations from above-ground parts of blighted trees in the vicinity of Beltsville, Maryland, and from samples sent in from diseased sweetgums in Delaware, Mississippi, Florida, Texas, Arkansas, and Louisiana. Altogether, isolations were made from 70 diseased trees.

Slivers of host tissues taken from fresh twigs and small branches of diseased trees, and from discolored areas of the sapwood, were plated on cornmeal, malt, and potato-dextrose agars. No one fungus was consistently isolated. The organisms isolated most frequently were species of Alternaria, Botrytis, Diplodia, Mortierella, Penicillium, Pestalotia, and Trichoderma. Over half of the platings, including those from brownish streaks in the sapwood, were sterile.

In 1952 two plots were established in pure stands of sweetgum, and during June, July, and August, 348 healthy trees were inoculated with pure cultures of the isolated fungi. The inoculated trees were approximately 20 years old, and about 1 to 6 inches in diameter at breast height. With a grafting knife an incision was made through the bark and a short distance into the wood. A piece of inoculum consisting of mycelia, substrate, and spores if present, was then introduced into the wound. A piece of moist cotton was placed over the wound, which was then wrapped with nursery grafting tape. Two to 15 inoculations were placed on the main stem and branches of each tree, depending on its size.

The trees have been examined four times since they were inoculated, the last examination being in September 1954. No sweetgum blight symptoms have appeared in the inoculated trees. From these and similar results of other workers, it appears unlikely that the blight is caused by fungi in the above ground portions of the tree.

Viruses

Many of the symptoms of sweetgum blight are characteristic of a systemic virus disease. All known virus diseases of woody plants are transmissible by one or more types of budding or grafting. In August 1953, one of these methods of transmission was used. Four patch-bark[2] grafts were placed on each of 100 healthy, vigorous sweetgum trees between 2 and 4 inches in diameter at breast height. The patch-bark method of grafting used consisted of replacing a piece of bark (1/2 x 1 inch in size) on a healthy tree with a similar-sized patch from a diseased tree. A patch-budding tool, known as the Jones Patch Budder, was used to cut the patch and the matrix into which it was inserted. The patch was firmly pressed into the matrix on the

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[2] Patches of living bark without buds.

healthy tree and tightly wrapped with nursery tape.

Ninety-six percent of the 360 bark-patches from diseased trees made successful unions when grafted into healthy trees. As a control, 40 bark-patches from healthy trees were grafted into healthy trees and 100 percent of these made successful unions. In September 1954, when the trees were last examined, there were no indications that the disease had been transmitted by patch-bark grafting, but the incubation period for some virus diseases is much longer than 13 months.

Nematodes

Soil and root samples collected from diseased and healthy sweetgum trees were examined for nematodes by the Section of Nematology, U. S. Department of Agriculture. In these samples many nematodes, most of them free-living or saprophytic forms, were found. In addition, a number of plant-parasitic species were found, but there was no apparent correlation between these nematodes and the disease symptoms.

Deficient or Excess Elements

On August 25, 1953, leaf samples were collected from healthy and diseased trees near Beltsville, Maryland, for chemical analyses to determine what elements, if any, are conspicuously low or high in the foliage of diseased trees. The samples consisted of 1) terminal leaves from primary shoots on main branches and 2) leaves from secondary shoots 24 inches or more back from the branch terminal. These were composite samples taken from three trees in each class. The results of analyses, performed by the Spectrochemical Laboratory, Horticultural Crops Research Branch, Beltsville, Maryland, are shown in Table 1.

Table 1. Amounts of certain elements in leaves collected from sweetgum trees showing different degrees of severity of the blight.

Tree condition	Elements - ppm*									
	Cu	Mn	Fe	Al	B	Na	P	Mg	Ca	K
Leaves collected from terminal ends of branches:										
Apparently healthy	11.0	435	170	510	39	100	1400	3800	7400	6500
Early symptoms	6.5	325	110	360	34	120	1400	2500	5100	6000
Advanced symptoms	6.3	308	110	280	45	120	1600	2900	6600	6500
Leaves collected 24 inches or more back from terminal ends of branches:										
Apparently healthy	9.7	450	200	540	43	150	1400	4100	9100	5500
Early symptoms	6.2	420	140	380	41	110	1600	2900	8600	7000
Advanced symptoms	8.3	520	210	370	66	85	1900	3600	9900	7000

*Each value is the mean for 3 samples of leaves.

There were no consistent differences in the concentration of the various elements in the leaves from healthy and diseased trees. Hence, it would seem that the blight disease of sweetgum is probably not caused by nutritional disorders. The analyses of variances of the data for the various elements indicated that only the concentration of aluminum was significantly different in the leaves of apparently healthy and diseased trees. Aluminum is not considered to be one of the essential elements.

Root Condition

Roots of 27 trees were excavated in 1953 to determine whether any relation existed between root condition and crown deterioration. Starting at the root collar at least two large roots of each tree were followed out, and several feet of fine roots were collected. At least 2,000 roots and rootlets from each tree sample were examined under a low-power dissecting microscope and classified as to whether they were living or dead. Starting with the smallest feeding roots

Table 2. Root mortality of sweetgum.

Condition of tree	Number trees examined	Type 1[a]		Type 2		Type 3		Type 4	
		Roots examined Number	Dead %	Roots examined Number	Dead %	Roots examined Number	Dead %	Roots examined Number	Dead %
Apparently healthy	12	30,131	14	12,649	10	2,779	10	72	0
Early symptoms	9	29,850	30	16,012	19	3,771	19	81	10
Advanced symptoms	6	19,835	59	6,309	47	1,931	44	59	15

[a] See text for types.

and working in toward the stem, the roots were typed as follows: 1) smallest feeding roots, 2) larger roots from which type 1 branch, 3) larger roots from which type 2 branch, and 4) larger roots from which type 3 branch. A summary of root mortality is presented in Table 2.

Table 2 indicates that there is a rather large difference between root mortality of diseased and apparently healthy sweetgum trees. For example, mortality of the smallest feeding rootlets of the nine trees exhibiting early symptoms of the blight was over twice that of the apparently healthy trees. However, additional information is needed on the rootlet condition of healthy vigorous sweetgum trees. Mortality of the smallest rootlets of the 12 apparently healthy sweetgum trees ranged from 4 to 27 percent. Some of the apparently healthy trees with a higher rootlet mortality may actually have been in the first stages of blight. It is entirely possible that death of the feeding roots may precede crown symptoms of the disease, the latter being simply a result of the reduced absorptive ability of the roots. No recognized pathogenic fungi were isolated from the roots of diseased trees even though repeated attempts were made.

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WEEDS AS IMPORTANT INTERIM HOSTS OF BROOMRAPE

Stephen Wilhelm[1] and Lee C. Benson[2]

The views that seed of broomrape, Orobanche ramosa L., may remain dormant in soils for as long as 13 years (2), and that it germinates only in response to stimulation by a preferred host such as tomato, tobacco, or hemp are of long standing. However, the lack of any evidence of successful wind dissemination and the apparent adverse effects of prolonged drying on seed survival raised some doubts as to the reported longevity of the dormant seed (3).

That marginal survival may occur on certain weeds is suggested in the paper of Durbin (1). However, the weed hosts reported were all found among parasitized tomato plants, which were assumed to have provided the stimulation needed for germination.

Recent searches (October-November 1954) showed broomrape growing in considerable abundance on weeds in harvested fields of cucumber and along dirt roads separating old sugar beet fields. These two crops are not hosts. Three principal weeds were involved, e.g., Solanum sarachoides Sendt., Amaranthus retroflexus L,, and Xanthium spinosum L. Growth of broomrape varying in age from new shoots just appearing above ground to shoots with mature seed capsules was present. The cucumber field had been planted to tomatoes in 1952, and the tomatoes were only lightly parasitized.

These observations leave no doubt that broomrape can attack and mature seed on certain weeds independent of an economic host, and they suggest that this mode of survival may be of importance in California. Also the fact that broomrape may appear above ground and mature seed late in the year after harvest of crops such as cucumber, sugar beet, and tomato is significant.

Destruction of weeds in harvested fields and along dirt roads in the general area infested by broomrape, together with dry fallow, may serve to check the pest.

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NOTES ON PLANT DISEASE INCIDENCE IN THE MATANUSKA AND TANANA VALLEYS, ALASKA - 1953 AND 1954

Charles E. Logsdon[1]

Bacterial ring rot (Corynebacterium sepedonicum) was epidemic in potatoes in both areas in 1953. Estimates of loss due to this disease and to complications following were 10 to 15 percent of the crop[2]. Higher than normal temperatures during the growing season seemed to contribute to the development of the epidemic[3]. Major complications following rot infection were rotting, heating, and sprouting leading to considerable bin pressure followed by the development of internal black spot.

Blackleg of potatoes was of minor importance in 1953, but was much more prevalent in 1954. The potato selection 10.42-2-46, inoculated with ring rot in experimental plots, failed to develop ring rot symptoms; but 20 percent of the plants developed blackleg (Erwinia phytophthora).

Most potato fields had relatively few virus-diseased plants although one field in the Tanana Valley was noted that had at least 50 percent of the plants infected with mild mosaic. The most important potato virus diseases in Alaska were mild mosaic and witches broom. In both 1953 and 1954 there were some giant hill plants and what appeared to be haywire.

Potato scab (Streptomyces scabies) was a serious problem in both areas. The scab organism, for the most part, seemed to be confined to localized areas in the fields, ranging in size from spots 10 to 15 feet in diameter to several acres. One field in the Matanuska Valley was so badly infested that potatoes were not planted in either 1953 or 1954 except for an experimental plot to determine the distribution of the scab organism. Even in this field the distribution of the scab was "spotty."

Fungus diseases of the growing potato plants were of minor importance in both years, although various fungus-induced rots caused considerable loss during the 1953-54 storage period. There was no late blight (Phytophthora infestans)in these areas in either year, and only one field was noted in 1953 in which there was any appreciable amount of early blight (Alternaria solani) on the foliage. Rhizoctonia (R. solani caused very little damage, and most of the plants examined were free from Rhizoctonia stem lesions. In spite of the lack of these stem lesions, many of the potato plants had a luxuriant growth of the perfect stage, Pellicularia filamentosa, on the lower parts of the stem. Whether this apparent anomaly was due to environmental conditions has not been determined.

Leak (? Pythium) caused some loss to potato growers in the Matanuska Valley in 1953 and was reported to have caused some loss in the Tanana Valley in 1954.

In 1953 there was blighting of potato blossoms and spotting of the leaves by Botrytis sp., but no evidence that the organism caused any other potato damage. Botrytis sp. also caused a stem rot and leaf spot of tomatoes, fruit rot of cucumbers, damping-off of lettuce, bulb rot of onion, and fruit rot of squash in storage. Considerable loss of carrots in storage also was associated with Botrytis infection although it was not determined if the Botrytis was a primary or a secondary invader.

Tomato leaf mold (Cladosporium fulvum) destroyed the entire tomato crop of one greenhouse in 1953, but was found that year in only one other greenhouse. The disease also occurred in 1954, but was not serious.

Lettuce growers in the Matanuska Valley sustained losses in several plantings in both years because of tipburn and slime.

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[1] Plant Pathologist, Agricultural Experiment Station, Palmer, Alaska

[2] Wilson, J. W. 1954. Alaska Market News. Vol. 4, No. 4. Apr. 20.

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NOTES ON SOME PLANT DISEASES IN TENNESSEE

E. L. Felix

Strawberry Powdery Mildew

Fragaria viridis (P.I. 202740) and cultivated strawberry were equally susceptible and Indian strawberry (Duchesnea indica) apparently immune to powdery mildew (presumably Sphaerotheca humuli) in the greenhouse during the summer and fall of 1954. Perithecia did not form. Parathion dust (1% active) appeared to check the mildew slightly, but failed to control it. A trace of mildew occurred also on Missionary in a garden in July, but disappeared soon afterward.

Downy Mildew of Turnip "Root"

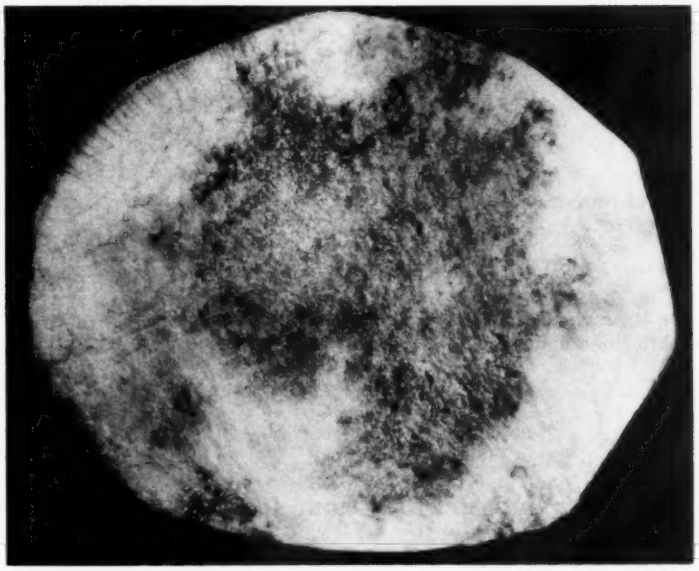

FIGURE 1. Slice of peeled turnip, showing internal discoloration and downy mildew fungus. Enlarged about 1 7/8 x.

One out of 20 Purple Top White Globe turnips, purchased in October, 1954, from a grocer and said to have been grown locally, showed extensive firm, brown internal discoloration (Fig. 1). Excellent sporulation of Peronospora parasitica occurred within 24 hours on moist incubation of slices, as reported by Gardner (Phytopath. 10: 321-322. 1920). Actually most of the turnip is enlarged stem and it was not determined whether the true root was infected also. No external symptom was observed.

Unusual Symptoms of Rhizoctonia Collar Rot of Tomato Seedlings

A sharply delimited collar rot, characterized by alternating dark-brown to reddish-brown and light-brown bands, appeared on a great many tomato seedlings in the greenhouse during the hot summer months of 1954 (Fig. 2). Symptoms were similar to those described and illustrated by Conover (Phytopath. 39: 950-951. 1949) on the branches of older tomato plants, and in fact later two older plants did have similar cankers on their branches. A Rhizoctonia resembling R. solani, with small sclerotia, was isolated consistently from the collar rot of the various tomato seedlings, but on inoculation during the winter produced the usual patternless type of damping-off on 116 out of 118 inoculated seedlings (variety Rutgers). Naturally infected fruit lying on the

FIGURE 2. Patterned collar rot of tomato seedlings. Approximately 3/4 natural size.

greenhouse bed at this time also lacked the usual zonation, altogether suggestive of temperature relations. Some tomato lines appeared to show considerable resistance to collar rot.

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SYNOPSIS OF RUSTS AND DOWNY AND POWDERY MILDEWS ON LEGUMINOUS CROPS IN ISRAEL

J. Palti and F. Nitzany

In the following tables free use has been made of the identifications of fungi pathogenic on legumes in Israel, as published by Rayss[1] and by Reichert[2] and their co-workers. The authors are also indebted to Professor I. Reichert for permitting them to use some of the data collected by the Division of Plant Pathology of the Agricultural Research Station, Rehovot, concerning the distribution of these fungi in Israel.

Table 1. The rust and mildew pathogens of legumes in Israel, their regional and seasonal distribution and their economic importance.

CROP Pathogen	Host parts affected	Regional distribution	Season of occurrence	Severity of loss caused
I. RUSTS				
CICER ARIETINUM, CHICKPEA				
Uromyces ciceris-arietini (Grogn.) Jacz.	Leaves	Rare	?	Slight
GLYCINE SOYA, SOYA BEAN				
Uromyces sp.	Leaves	Rare	?	?
LATHYRUS SATIVUS, CYPRUS VETCH				
Uromyces sp.	Leaves, stalks	?	April-June	Slight to moderate
LENS ESCULENTA, LENTIL				
U. fabae (Pers.) DBy.	Leaves	Coastal belt, low valleys	March-June	Slight
LUPINUS SATIVUS, LUPIN				
? U. renovatus Syd.	Leaves	Coastal belt	December-February	Slight
MEDICAGO SATIVA, ALFALFA				
U. striatus Schroet.	Leaves, stalks	Ubiquitous	All seasons	Slight to moderate
PHASEOLUS VULGARIS, BEAN				
U. phaseoli (Pers.) Wint.	Leaves, stalks, pods	Coastal belt, less in Galilee and Jerusalem region	June-December	Moderate
PISUM ARVENSE, P. SATIVUM, PEAS				
U. pisi (Pers.) DBy.	Leaves	Ubiquitous	November-May	Moderate

[1] Rayss, T. Palestine Jour. Bot., Jerusalem Ser., 3(3): 151-166, 1945; ibid. 4(2): 59-76, 1947; "Uredineana" 3: 154-221, 1951. Savulescu, Th., and T. Rayss, "Hommage au Prof. Teodoresco" pp. 163-175. 1937.

[2] Reichert, I. Int. Bull. Plant Prot., Rome, 13(9): 204-210, 1939; ibid. 13(10): 225-240, 1939.

Table 1. (Continued).

CROP Pathogen	Host parts affected	Regional distribution	Season of occurrence	Severity of loss caused
TRIFOLIUM ALEXANDRINUM, BERSEEM CLOVER				
U. trifolii (Hedw. f.) Lév.	Leaves, stalks	Ubiquitous	November-July	Moderate
TRIGONELLA FOENUM-GRAECUM, FENUGREEK				
? U. anthyllidis Schroet. f. trigonellae Rayss	Leaves	Ubiquitous	April-June	Slight to moderate
VICIA FABA, BROAD BEAN				
U. fabae (Pers.) DBy.	Leaves, stalks, pods	Ubiquitous	December-May	Very severe
VICIA SATIVA, VETCH V. ATROPURPUREA, PURPLE VETCH				
U. fabae (Pers.) DBy.	Leaves, stalks	Ubiquitous	March-June	Slight to moderate
VIGNA SINENSIS, COWPEA				
U. phaseoli var. vignae (Barcl.) Arth.	Leaves	?	Spring	Moderate
II. POWDERY MILDEWS[a]				
ARACHIS HYPOGAEA, GROUNDNUT				
Oidium sp.	Leaves	Rare	July-October	Slight
LATHYRUS SATIVUS, CYPRUS VETCH				
Oidium sp.	Leaves	?	May-June	Slight
LUPINUS SATIVUS, LUPIN				
Oidium sp.	Leaves	Coastal belt	January-April	Slight
MEDICAGO SATIVA, ALFALFA				
Oidium sp.	Leaves	Coastal belt, low valleys	All seasons	Slight
Oidiopsis taruica (Lév.) Salm.	Leaves	Negeb and low valleys	September-April	Slight
PHASEOLUS AUREUS				
Oidium sp.	Leaves	?	May-June	Slight
PHASEOLUS VULGARIS, BEAN				
Oidium sp.	Leaves	Rare	March-November	Slight
PISUM ARVENSE, P. SATIVUM, PEAS				
Oidium sp.	Leaves, stalks, pods	Ubiquitous	November-June (especially March-May)	Severe
TRIFOLIUM ALEXANDRINUM, BERSEEM CLOVER				
Oidium sp.	Leaves	Ubiquitous	March-July	Slight to moderate

Table 1. (Continued).

CROP Pathogen	Host parts affected	Regional distribution	Season of occurrence	Severity of loss caused
TRIGONELLA FOENUM-GRAECUM, FENUGREEK				
Oidiopsis taurica (Lév.) Salm.	Leaves, stalks, pods	Probably ubiquitous	April-June	Severe
VICIA SATIVA, VETCH, V. ATROPURPUREA, PURPLE VETCH				
Oidium sp.	Leaves, stalks	Ubiquitous	April-June	Severe
III. DOWNY MILDEWS				
LATHYRUS SATIVUS, CYPRUS VETCH				
Peronospora sp.	Leaves	Rare	April-May	Slight
MEDICAGO SATIVA, ALFALFA				
P. aestivalis Syd.	Leaves	Coastal belt	All seasons, especially March-July	Slight to moderate
PISUM ARVENSE, P. SATIVUM, PEAS				
P. pisi	Leaves	Coastal belt, Esdraelon Valley North and West Negeb	February-May	Moderate
TRIFOLIUM ALEXANDRINUM, BERSEEM CLOVER				
Peronospora sp.	Leaves	Coastal belt	December-February	Slight
TRIGONELLA FOENUM-GRAECUM, FENUGREEK				
P. trigonellae (?)	Leaves	Rare	February-April	Moderate to Severe
VICIA FABA, BROAD BEAN				
Peronospora sp.	Leaves	Coastal belt	December-April	Slight
VICIA SATIVA, VETCH, V. ATROPURPUREA, PURPLE VETCH				
P. viciae-sativae (?)	Leaves	Coastal belt, Esdraelon Valley	January-April	Slight

TEL-AVIV, ISRAEL

[a] No perithecia have so far been found on any of the powdery mildews collected on leguminous crops in Israel.

PLANT DISEASE LOSS ESTIMATES IN 1954 FOR TENNESSEE

J. O. Andes[1]

Notwithstanding the limitations of plant disease loss estimates based on observations and opinion there is a definite need for a yearly approximation, and in order to reach a more nearly correct appraisal this compilation was made from estimates of the various workers in plant pathology and certain crop fields. In the case of cotton and tobacco data were kept during the season, otherwise the estimates were made from observations. The popular impression is that due to the extreme drought of 1954 plant disease losses were nil. On the contrary, however, the overall State loss was significant and in some specific cases quite heavy.

Certain generalities seem warranted that should have a bearing on planning for future work. Irrigation has been resorted to in an unprecedented manner and present indications are that it will be extensively employed in the future. It is noteworthy that in some cases, particularly with vegetable crops, during the extreme drought irrigation brought about serious losses from plant diseases. On the other hand, the wider use of resistant varieties, notably in black shank of tobacco, was thought to account for the lower losses to a measurable extent.

Table 1. Estimated loss from diseases of fruits and vegetables in Tennessee, 1954.

CROP Disease	Occurrence	Percent loss		Remarks
		State	Local	
APPLE				
Scab, Venturia inaequalis	General	Trace	5	
Cedar rusts, Gymnosporangium spp.	General	Trace	2	Intensity varied with location, primarily due to lowered vigor of tree.
Fire blight, Erwinia amylovora	General	2	10	
PEAR				
Fire blight, Erwinia amylovora	General	3	10	
PEACHES				
Brown rot, Monilinia fructicola	General	1	3	
Scab, Cladosporium carpophilum	General	2	5	Primarily reduction in quality.
STRAWBERRY				
Fruit rot, Botrytis cinerea	General	2	10	
Virus	General	3	5	
TOMATO				
Wilt, Fusarium oxysporum f. lycopersici	Local	2	10	
Nematodes	General	5	20	
Fruit rots, Phytophthora parasitica	Local	Trace	10	Principally under irrigation.
Southern blight, Sclerotium rolfsii	Local	1	20	
SWEETPOTATO				
Stem rot, Fusarium oxysporum f. batatas	General	1	5	Mainly in West Tennessee.

[1] Compiled from estimates provided by J. M. Epps, D. H. Latham, R. P. Mullett, E. L. Felix, and Gilbert Rhodes.

Table 2. Estimated loss from diseases of field crops in Tennessee, 1954.

CROP Disease	Occurrence	Percent reduction Statewide	Percent reduction Local	Remarks
SOYBEANS				
Damping-off, Rhizoctonia solani	General	Trace	10	Affects stand, probably not reflected in yields.
Leafspots, bacterial	General	Trace	Trace	
CORN				
Ear, stalk, root rots, and seedling blight, Diplodia spp. and Fusarium spp.	General	4-5	10-15	
Leafspot, Helminthosporium spp.	General	Trace	4-5	
WHEAT				
Loose smut, Ustilago tritici	General	Trace	2-3	
Leaf rust, Puccinia rubigo-vera tritici	General	Trace	3-4	
Stem rust, P. graminis tritici	Local	--	Trace	
Scab, Gibberella and Fusarium spp.	General	Trace	2	
Glume blotch, Septoria nodorum	General	Trace	Trace	
BARLEY				
Spot blotch, Helminthosporium sativum	General	1	3	
Scald, Rhynchosporium secalis	General	2	25	Especially severe on Colonial
Loose smut, Ustilago nuda	General	1	8	
Mildew, Erysiphe graminis	General	1	3	
OATS				
Smuts, Ustilago spp.	General	Trace		
Crown rust, Puccinia coronata	General	Trace		
Mildew, powdery, Erysiphe graminis avenae	General	Trace		
COTTON				
Fusarium wilt, Fusarium oxysporum f. vasinfectum	General	0.5		
Verticillium wilt, Verticillium albo-atrum		0.2		
Seedling diseases		0.5		
Freezing		1.5		

Table 3. Estimated loss from diseases of tobacco in Tennessee, 1954.

Disease	Occurrence	Percent reduction		Remarks
		Statewide	Local	
Wildfire and blackfire *Pseudomonas tabaci* *Pseudomonas angulata*	General, mainly in plant beds	2-3	2-3	Actual loss in field small--reduction due to delay in setting, resulting in lower yield and quality.
Fusarium wilt *Fusarium oxysporum* f. *nicotianae*	Localized	Trace	1	
Frenching	General	Trace	1-2	
Blue mold *Peronospora tabacina*	Localized	None	---	
Frog eye *Cercospora nicotianae*	General	Trace	5	Came late in season. Losses incurred only under irrigation.
Black shank *Phytophthora parasitica* var. *nicotianae*	General	1	5	Spreading to 3 new counties.
Mosaic (virus)	General	1	3	More pronounced on dark than burley.
Anthracnose *Colletotrichum* spp.	Local	---	Trace	In plant beds only.
Nematodes	General	Indeterminate	---	

Overall State estimate for all tobacco diseases 3 percent.

UNIVERSITY OF TENNESSEE, KNOXVILLE

UNUSUAL RECORDS OF PLANT DISEASE OCCURRENCE

GIBBERELLA FUJIKUROI THE CAUSE OF BUD ROT OF CORN IN PUERTO RICO

By R. H. Segall and J. Vélez Fortuño[1]

Experimental corn hybrids grown at Isabela, Puerto Rico last fall were destroyed by a serious outbreak of bud rot caused by Gibberella fujikuroi (Fusarium moniliforme). The corn was planted on August 18, 1954, and symptoms were first noted on September 11. Between September 4 and 7, 4 1/2 inches of rain fell. A susceptible line planted 2 weeks after the earlier plants became infected remained healthy and gave a normal yield.

Symptoms of the disease consisted of rotting of buds and of leaves as they emerged from the buds. The leaves then turned yellow and died. The diseased plants remained small, 2 to 3 feet high, and failed to tassel. Macro- and micro-spores of Fusarium were found in the rotting buds and the causal organism isolated. The field containing the diseased corn was adjacent to sugar cane which had symptoms of "pokkah bong disease" also caused by Gibberella fujikuroi.

The last serious outbreak of bud rot of corn reported in Puerto Rico occurred in June 1942, also at Isabela[2]. At that time 3.61 inches of rain fell in 1 week, approximately 2 weeks after planting and approximately 1 week before symptoms were first reported.

AGRICULTURAL EXPERIMENT STATION OF THE UNIVERSITY OF PUERTO RICO

[1] Associate Plant Pathologist and Associate Plant Breeder, respectively, of the Agricultural Experiment Station, University of Puerto Rico, Río Piedras, Puerto Rico.

[2] Nolla, J. A. B. 1944. Annual report for the fiscal year 1942-43 of the University of Puerto Rico - Agricultural Experiment Station. pp. 37-38.

BOTRYTIS ROT OF CALADIUM TUBERS

By H. N. Hansen and Robert V. Bega[1]

About 20 percent of a shipment of caladium tubers (Caladium bicolor Vent.) imported into California from Florida were found to have a soft rot somewhat similar to that caused by Erwinia carotovora (L. R. Jones) Holland. The entire shipment was returned.

Symptoms of the disease are undetectable externally unless the softened infected areas of the tuber are touched. When the tubers are cut, the internal diseased tissues appear grey-brown in color and pulpy or pithy in texture, with a distinct zone between sound and diseased areas. No odor is detectable. There may be one, two, or more infection sites on one tuber.

Isolations from these infected areas yielded a species of Botrytis which appears to be B. (Botryotinia) ricini (Godfrey) Whet. This organism is known to be an important pathogen on castor bean (Ricinus communis L.). The disease on castor bean was first reported and described by Stevens[2, 3], in Florida, and later by Godfrey[4, 5], who worked with and described the causal organism and further described the disease.

Sound tubers of three varieties of caladium were surface-sterilized in 10% Clorox, inoculated with the fungus, and then placed in moist chambers and held at room temperature. After approximately 1 week's time the symptoms were reproduced. We believe this to be the first report of a disease of caladium caused by this organism.

When and how invasion occurs in the field remains to be investigated, but growing caladiums in land previously cropped to castor beans would at this time appear inadvisable.

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[1] Respectively, Professor of Plant Pathology, and Research Assistant in Department of Plant Pathology, University of California, Berkeley 4, California.

[2] Stevens, H. E. 1918. Gray mold of castor beans. Florida Agr. Exp. Sta. Press Bul. 296, 2pp.

[3] Stevens, H. E. 1918. Gray mold of castor bean. Florida Grower 18: 6.

[4] Godfrey, G. H. 1919. Sclerotinia ricini n. sp., parasitic on the castor bean (Ricinus communis). Phytopath. 9: 565, 567.

[5] Godfrey, G. H. 1923. Gray mold of castor bean. Jour. Agr. Res. 22: 679-716.

DIGITALIS DISEASES IN WISCONSIN IN 1954

By Robert W. Fulton and Earl K. Wade

A small acreage of Digitalis lanata Ehrh. has been grown commercially in Wisconsin since World War II with only minor losses from disease. During the 1954 season, however, two diseases appeared which were destructive and caused the growers concern.

One of these was anthracnose (Colletotrichum fuscum Laub.) which was present in every field seen. The severity ranged from a few scattered leaf lesions to complete killing of appreciable numbers of plants in some fields by October. One field planted to the species D. purpurea L., in the same area, showed only a small amount of anthracnose. Thomas[1] reported that this species does not become as severely infected with anthracnose as does D. lanata.

The other disease was a severe, malforming mosaic caused by tobacco mosaic virus. This was also present in every field seen. Sample counts in the most severely affected field showed over 50 percent of the plants infected. Some of these had mosaic patterns on all leaves, including the oldest, indicating early infection. On others symptoms were present only on the youngest leaves.

Inoculations from 12 plants from the most severely affected field yielded, from 11, only tobacco mosaic virus. Most mosaic plants tested from other fields also contained tobacco mosaic virus. A few contained what was apparently cucumber mosaic virus, and one yielded a virus producing ring-spotting on tobacco.

Since most Digitalis is grown by tobacco growers the presence of tobacco mosaic virus is not surprising. Limited surveys in the past, however, have shown only a low incidence of virus diseases. As far as we are aware the natural occurrence of this virus on Digitalis lanata has not been reported previously.

WISCONSIN AGRICULTURAL EXPERIMENT STATION, AND FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE

[1] Thomas, C. A. 1951. Anthracnose of Digitalis. Phytopathology 41: 997-1000.

THE PLANT QUARANTINE PROBLEM: BOOK REVIEW

"THE PLANT QUARANTINE PROBLEM: A General Review of the Biological, Legal, Administrative and Public Relations of Plant Quarantines with Special Reference to the United States Situation", by W. A. McCubbin. 255 pp. 1954. Ejnar Munksgaard, Copenhagen, Denmark, publisher. American distributors: The Chronica Botanica Co., Waltham, 54, Mass., U. S. A. Price: Da. kr. 32, -, sh. 33/9, $4.80.

This thorough examination, the first complete one, of the principles, management, and effects of plant quarantines is written by a plant pathologist whose career included more than thirty year's experience in the plant quarantine field. It is intended not so much for plant quarantine officials, who the author says do not need it, as for the citizen of any calling who may need or just want to know about quarantines. However, the book could serve as an excellent refresher course for quarantine officials. Pathologists and entomologists not in the quarantine field also could read it for a better understanding of their professional contributions to the success of quarantines, as well as of their private responsibilities as citizens affected by quarantine restrictions.

The nontechnical person who reads it will acquire much more than merely information about quarantines. The clear explanation as to why plant pests (including pathogens and insects) are important and how much trouble and money they cost gives a foundation for appreciation of the biological hazards of agriculture, about which the average person has only the vaguest notion. The author uses everyday experience as the starting point for his explanation. He breaks losses down into individual units that make the billions of dollars annual estimates a matter of personal expense rather than abstract finance, and shows that everybody is affected.

This is the necessary minimum information for understanding why it is important to prevent introduction of destructive plant pests from outside the country or to keep them from spreading within it. On this foundation the author describes quarantine procedure. He shows clearly that quarantines are not arbitrary but on the contrary are decided upon only after all the circumstances in the particular situation are evaluated and everybody directly affected is notified and consulted. Some of the important items considered in establishing quarantines are the destructiveness of the pest, the value of the crop or crops affected, especially as a basic food or fiber crop, the cost of preventing introduction or spread vs. the cost of control if the pest should become established, and the likelihood of success, as judged from biological and geographical

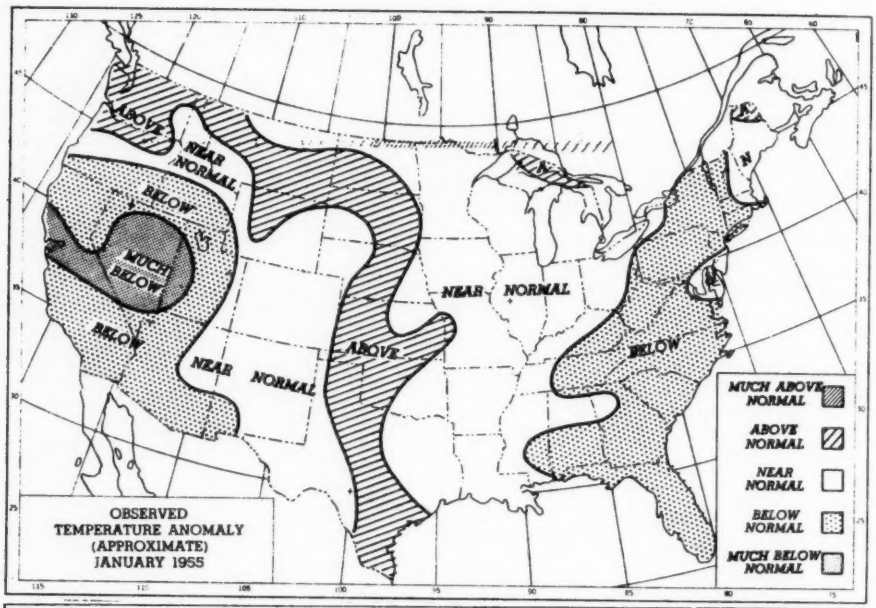
factors, probable public support, and timeliness of action. In discussing the evaluation of the probable benefits of a quarantine the author notes the lack of reliable bases for appraising damage to crops caused by pests.

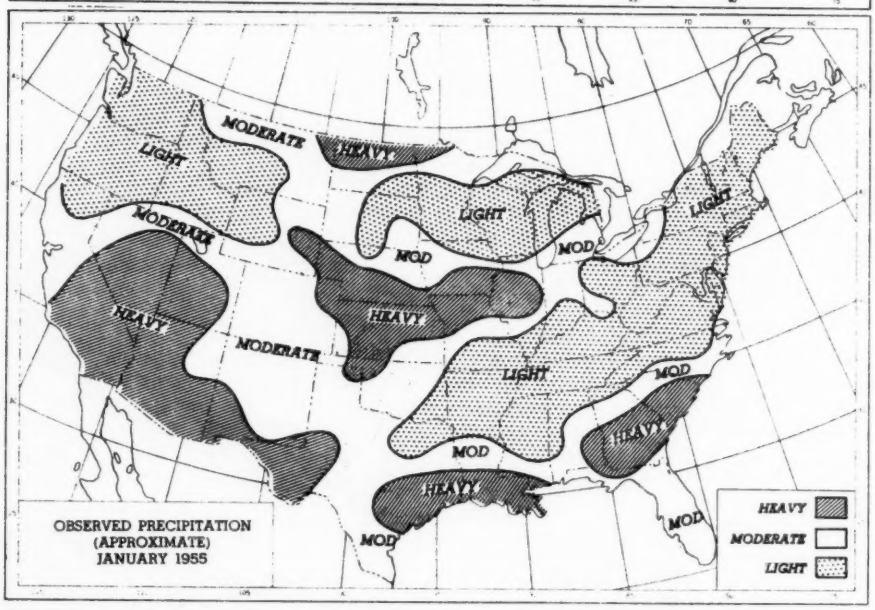
Administrative and legal relations are usually rather dull topics but the author has managed to make them interesting. In particular, he shows that a certain amount of "red tape", i. e., record keeping, which always arouses antagonism, is nevertheless indispensable for the protection of everybody in a quarantine action.

Quarantine problems are illustrated by a detailed account of the potato golden nematode quarantine. The differences and relations between Federal and State quarantines are explained. An appendix contains a historical summary of Federal plant quarantines and explores the reasons for success or failure of each one.

The author concludes that long-time quarantine planning is desirable in a quarantine-minded world, but that the present "zig-zag" between weak and effective action is much more likely to continue.

This review does not adequately indicate the thoroughness with which all phases of the quarantine problem are investigated, nor the clarity with which they are explained. The simple interesting style is one of the features that make the book admirably suited to its stated purpose and a distinct contribution to plant pathological literature. -- P. R. M. and J. I. W.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

MANUSCRIPTS FOR THE PLANT DISEASE REPORTER

(See also PDR 35: 121-122; 36: 214, 494)

(1) GENERAL: The Reporter page measures 9 inches long with the heading or 8 3/4 inches for the text part, by 6 inches wide. The copy is typed on a larger page, 11 1/4 inches of text or 12 inches overall in length by 8 inches in width, and reduced 25 percent in the photographic process of reproduction. Illustrations or tables larger in either dimension will take a correspondingly greater reduction. Only one size of type is available for text, footnotes, or tables.

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(3) ABSTRACTS are requested for all except very short articles. (PDR 38: 316).

(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so. (PDR 36: 494).

(5) LITERATURE REFERENCES should be given in alphabetical order and numbered for citation in the text. We follow the AIBS suggestion of placing the year of publication after the author's name. Please check your titles carefully since we cannot do it for you. Be sure that text citations and bibliography agree; that foreign-language references are correct; that number or month is cited for periodicals that are not paged consecutively throughout the volume.

(6) NAMES OF FUNGICIDES should be given according to the suggestions that will be published in Phytopathology before long, and later repeated in the Reporter. In the meantime see PDR 35: 122.

(7) ILLUSTRATIONS should be sent to us unmounted. To prevent mistakes, write figure numbers on the back, and mark the top of each print when necessary. A sketch can show a preferred arrangement but please keep in mind page size, shape, and standard reduction (see above under General), and remember that figure titles and legends are part of the page. Lettering should be clear and large enough to be legible after reducing. Drawings, maps, and graphs can be photographs or originals, but should be finished and ready for reproduction, not just sketches.

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